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Triclopyr - Revised Human Health and Ecological Risk Assessments Final Report

Prepared for:

USDA, Forest Service

Forest Health Protection

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Supplement 2: Triclopyr BEE (Garlon 4) -Worksheets for Human Health and Ecological Risk Assessments, SERA WPWS 01-43-15-02c (WordPerfect) and SERA EXWS 01-43-15-02c (EXCEL) dated February 18, 2003.

ATTACHMENTS

Attachment 1: Bibliography of Citations Encountered.

Attachment 2: Documentation for Worksheets Version 2.04 - Human Health and Ecological Risk Assessments, SERA WSD 01-2.04 dated February 25, 2003.

Attachment 3: Documentation for the Use of GLEAMS and Auxiliary Programs, SERA AT 2000-01d, dated October 12, 2001.

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ACRONYMS, ABBREVIATIONS, AND SYMBOLS

a.e.	acid equivalents
a.i.	active ingredient
AEL	adverse-effect level
ACGIH	American Conference of Governmental Industrial Hygienists
ATSDR	Agency for Toxic Substances and Disease Registry
BCF	bioconcentration factor
BEE	butoxyethyl ester
BUN	blood urea nitrogen
bw	body weight
CAS	Chemical Abstracts Service
CBI	confidential business information
C.I.	confidence interval
cm	centimeter
DRES	Dietary Risk Evaluation System
EC_{50}	concentration causing 50% inhibition of a process
EC_{100}	concentration causing complete inhibition of a process
EDTA	ethylenediaminetetra acetic acid
EIS	environmental impact statement
F	female
F_1	first filial generation
FH	Forest Health
FIFRA	Federal Insecticide, Fungicide, and Rodenticide Act
FS	Forest Service
FQPA	Food Quality Protection Act
g	gram
GC	gas chromatography
HQ	hazard quotient
IARC	International Agency for Research on Cancer
IRIS	Integrated Risk Information System
k_a	absorption coefficient
k_e	elimination coefficient
kg	kilogram
$K_{o/c}$	organic carbon partition coefficient
K_{ow}	octanol-water partition coefficient
K_p	skin permeability coefficient
L	liter
lb	pound
LC_{50}	lethal concentration, 50% mortality
LD_{50}	lethal dose, 50% mortality
LD_{95}	lethal dose, 95% mortality
LOAEL	lowest-observed-adverse-effect level

LOD	limit of detection

ACRONYMS, ABBREVIATIONS, AND SYMBOLS (*continued*)

m	meter
M	male
mg	milligram
mg/kg/day	milligrams of agent per kilogram of body weight per day
mL	milliliter
MS	mass spectrometry
MW	molecular weight
MOS	margin of safety
MRID	master record identification number
MSDS	material safety data sheet
NCI	National Cancer Institute
NOAEL	no-observed-adverse-effect level
NOEC	no-observed-effect concentration
NOEL	no-observed-effect level
NRC	National Research Council
OPP	Office of Pesticide Programs
pKa	acid dissociation constant
ppm	parts per million
PSP	phenolsulfonphthalein
RBC	red blood cells
RED	registration eligibility decision
RfD	reference dose
RTU	ready to use
TBEE	triclopyr butoxyethyl ester
TCDD	2,3,7,8-tetrachlorodibenzo-p-dioxin
TCP	3,5,6-trichloro-2-pyridinol
TEA	triethylamine
UF	uncertainty factor
U.S.	United States
U.S. EPA	U.S. Environmental Protection Agency
USDA	United States Department of Agriculture
USGS	United States Geological Survey
WCR	water contamination rate
>	greater than
≥	greater than or equal to
<	less than
≤	less than or equal to
=	equal to
≃	approximately equal to

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	1.8C°+32
centimeters	inches	0.3937
cubic meters (m^3)	liters (L)	1,000
Fahrenheit	centigrade	0.556F°-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
hectares (ha)	square meters	10,000
inches (in)	centimeters (cm)	2.540
kilograms (kg)	ounces, (oz)	35.274
kilograms (kg)	pounds, (lb)	2.2046
kilograms per hectare (hg/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 ($\mu g/cm^2$)	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

INTRODUCTION

This document provides risk assessments for human health effects and ecological effects to support an assessment of the environmental consequences of using triclopyr in Forest Service programs. The USDA Forest Service uses the herbicide, triclopyr, in its vegetation management programs. Five commercial formulations of triclopyr, either as the triethylamine (TEA) salt or the butoxyethyl ester (BEE) are currently registered for forestry applications and are covered in this risk assessment. An additional formulation of the TEA salt of triclopyr has been labeled for aquatic weed control and this use is also considered in the current risk assessment.

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

Almost no risk estimate presented in this document is given as a single number. Instead, risk is expressed as a central estimate and a range, which is sometimes very large. Because of the need to encompass many different types of exposure as well as the need to express the uncertainties in the assessment, this risk assessment involves numerous calculations. Most of the calculations are relatively simple, and the very simple calculations are included in the body of the document. Some of the calculations, however, are cumbersome. For those calculations, a set of worksheets is included as an attachment to the risk assessment. The worksheets provide the detail for the estimates cited in the body of the document. two versions of the worksheets are available: one in a word processing format and one in a spreadsheet format. The worksheets that are in the spreadsheet format are used only as a check of the worksheets that are in the word processing format. Both sets of worksheets are provided this risk assessment. Documentation for the use of these worksheets is also provided in a separate document that accompanies this risk assessment.

PROGRAM DESCRIPTION

Triclopyr is a herbicide that mimics auxin, a plant growth hormone, thus disrupting the normal growth and viability of plants. Triclopyr is used in Forest Service programs primarily for wildlife habitat improvement, noxious weed control, conifer or hardwood release, and site preparation, with other minor uses including rights-of-way management, hardwood control, facilities maintenance, and seed orchard protection. Two forms of triclopyr are used commercially as herbicides: the triethylamine salt and the butoxyethyl ester. Currently, there are 5 commercial formulations of triclopyr that are registered for forestry applications: Garlon 3A, Garlon 4, Forestry Garlon 4, Pathfinder II, and Remedy RTU. Garlon 3A contains the triethylamine salt of the triclopyr and inert ingredients and requires the use of a non-ionic surfactant. In addition to triethylamine, Garlon 3A contains EDTA, a common chelating agent, and ethanol. Garlon 3A, marketed as Renovate 3, has recently been labeled for aquatic weed

control. The other commercial formulations contain the butoxyethyl ester of triclopyr, often referred to as triclopyr-BEE. Garlon 4 and Forestry Garlon 4 both contain kerosene and proprietary surfactants. For this risk assessment and based on recent Forest Service applications, the average application rate is taken as 1 lb a.e./acre with a range of 0.05 lb a.e./acre to 10 lbs a.e./acre. The total annual use of triclopyr by the Forest Service for 2001 is about 7,700 lbs, which is about 4 percent of the agricultural use. Nonetheless, the agricultural uses of triclopyr are rather localized and, in some regions, the use of triclopyr in Forest Service programs may be a substantial source of triclopyr residues in the environment.

HUMAN HEALTH RISK ASSESSMENT

Hazard Identification **–** Studies regarding histopathology and clinical chemistry data on triclopyr suggest that the liver and kidney are the primary target organs. Like most weak acids, triclopyr is excreted primarily in the kidney by an active transport process. The dermal absorption of triclopyr BEE has been measured *in vitro* using flow-through diffusion cells with skin from rats and humans. In addition, an *in vivo* pharmacokinetics study involving oral and dermal exposure to triclopyr is available using human volunteers. Like any chemical, triclopyr at sufficiently high exposure levels can cause toxic effects, including death. Nonetheless, triclopyr has a low order of acute lethal potency. As large number of subchronic and chronic toxicity studies are available on triclopyr. All studies were submitted to U.S. EPA/OPP to the support the registration of triclopyr. Full copies of all studies were obtained and reviewed as part of the current risk assessment. There is no information suggesting that triclopyr causes direct adverse effects on the nervous system, endocrine system, or immune function. At doses which do not cause maternal toxicity, there is not apparent concern for either reproductive or teratogenic effects. At substantially higher doses that are maternally toxic, triclopyr has been shown to result in birth defects. Most of abnormalities have been indicative of delayed growth and have been associated with maternal toxicity. Standard bioassays for carcinogenicity have been 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 – 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 has reviewed these studies and determined that the evidence for carcinogenicity is marginal and has not recommended as 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. TCP is of concern to this risk assessment both because it is a metabolite of triclopyr and because the aggregate risks of exposure to TCP from the breakdown of both triclopyr and chlorpyrifos must be considered. While there is no indication that the general exposures to TCP from the use of triclopyr and chlorpyrifos will result in harmful levels of exposure, this risk assessment does

specifically include a consideration of such exposures that may result from specific program activities in the use of triclopyr and chlorpyrifos in forestry applications.

Exposure Assessment – Two types of exposure assessments are considered: general and accidental/incidental. The term *general* exposure assessment is used to designate those exposures that involve estimates of absorbed dose based on the handling of a specified amount of a chemical during specific types of applications. The accidental/incidental exposure scenarios involve specific types of events that could occur during any type of application. Based on analyses of several different pesticides using a variety of application methods, default exposure rates are estimated for three different types of applications: directed foliar (backpack), boom spray (hydraulic ground spray), and aerial. These exposure estimates are consistent with and supported by four worker exposure studies involving triclopyr applications.

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

Dose-response Assessment – Generally, the dose-response assessments used in Forest Service risk assessments adopt RfDs proposed by the U.S. EPA as indices of acceptable exposure. An RfD is basically defined as a level of exposure that will not result in any adverse effects in any individual. The U.S. EPA RfDs 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 U.S. EPA recommends a chronic RfD of 0.05 mg/kg/day. This chronic RfD is based on the two-generation reproduction study in rats in which degeneration of renal proximal tubules were noted in adult animals at a dose of 25 mg/kg/day but not at 5 mg/kg/day. The 5 mg/kg/day NOAEL dose was divided by 100, a factor of 10 to account for uncertainties in species-to-species extrapolation and another factor of 10 to encompass sensitive individuals in the population. Thus, the resulting RfD is 0.05 mg/kg/day. Under the Food Quality Protection Act (FQPA), the U.S. EPA is required to evaluate whether or not an additional uncertainty factor is required for the protection of children. Because the parental NOAEL for reproduction studies are below any

adverse reproductive effects, the U.S. EPA has determined that no additional FQPA uncertainty factor is required. The U.S. EPA has recommended an acute RfD for triclopyr of 1 mg/kg/day for the general population. This is based on the NOAEL of 100 mg/kg/day from at teratogenicity study (i.e., a study test the potential for the development of birth defects). This acute RfD is not applicable to females between the ages of 13-50 years – i.e., females of child bearing age. For these individuals, the acute RfD is set at 0.05 mg/kg/day, equivalent to the chronic RfD.

The U.S. EPA has not derived a formal RfD for TCP, the metabolite of triclopyr. For the current risk assessment, the risk values used for risk characterization are identical to the most recent and conservative risk values proposed by U.S. EPA: 0.025 mg/kg/day for acute exposures and 0.012 mg/kg/day for chronic exposures. The acute value is based on a developmental toxicity study in rabbits with NOAEL of 25 mg/kg/day and an uncertainty factor of 1000. The chronic risk value is based on a 12 mg/kg/day NOAEL, also using an uncertainty factor of 1000.

Risk Characterization **–** There is no indication that workers will be subject to hazardous levels of triclopyr at the typical application rate of 1 lb/acre and under typical exposure conditions. Nonetheless, at the upper range of exposures, all application methods exceed the level of concern based on the chronic RfD but not the acute RfD. Thus, for workers who may apply triclopyr repeatedly over a period of several weeks or longer, it is important to ensure that work practices involve reasonably protective procedures to avoid the upper extremes of potential exposure. At higher application rates, particularly rates that approach the maximum application rate of 10 lbs/acre, measures should be taken to limit exposure. These measures would need to be developed on a case-by-case basis depending on the specific application rates that are used and the type of the applications that are employed.

For members of the general public, the risk characterization is thus relatively unambiguous at the typical application rate of 1 lb/acre: based on the available information and under the foreseeable conditions of exposure, there is no route of exposure or exposure scenario suggesting that the general public will be at risk from longer-term exposure to triclopyr. Even at the maximum projected application rate of 10 lbs/acre, the only longer-term scenario that exceeds the level of concern is the consumption of contaminated fruit This is a standard scenario used in all Forest Service risk assessments and is extremely conservative – i.e., it assumes that fruit that has been directly sprayed is harvested and consumed for a prolonged period of time and that the contaminated fruit accounts for 100% of the individuals consumption of fruit. Under these extreme conditions, the level of concern (a hazard quotient of unity) is exceeded by a factor of 5 at the upper range but not the central estimate of exposure. Several acute exposures also lead to hazard quotients that are above the level of concern at the upper range of exposure. Two dermal exposures to triclopyr BEE – i.e., accidental spray of a woman over the lower legs as well as dermal contact with contaminated vegetation by a woman – exceed the level of concern at the central estimate of exposure. The use of the highest application under consideration – i.e., 10 lbs/acre – alters the risk characterization for acute exposures terms of dermal exposures and the spill into a pond. At an application rate of 10 lbs/acre, both triclopyr BEE and triclopyr TEA formulations would exceed the level of concern for all dermal exposure scenarios at the upper

range of exposure as well as some central estimates of exposure. Again, all of these dermal exposure assessments are extremely conservative and designed to identify which possible types of exposure would be most hazardous. For triclopyr, such scenarios include dermal contact and accidental spills into water.

The U.S. EPA has conducted extensive analyses of dietary exposure to TCP from the use of triclopyr as well as the aggregate risks from exposure to TCP from the use of both triclopyr and chlorpyrifos. While these dietary exposures appear to be substantially below a level of concern, the risk assessment by EPA does not specifically address concerns for contamination of water with TCP as a soil metabolite of triclopyr and chlorpyrifos. As part of the current risk assessment, exposures to TCP based on modeling of water contamination from the application of both triclopyr and chlorpyrifos indicate that the peak exposure to TCP in water is below the concentration associated with the chronic risk value for TCP. Thus, there is no basis for asserting that the use of triclopyr with or without the use of chlorpyrifos will result in hazardous exposures of humans to TCP.

ECOLOGICAL RISK ASSESSMENT

Hazard Identification **–** An assessment of the potential toxic hazards associated with the exposures of wildlife mammalian species to triclopyr is based on the same studies on experimental mammals that are used in the human health risk assessment. Although triclopyr causes developmental effects only at doses that cause maternal toxicity, reproductive effects are obviously an endpoint of concern to both the human health and ecological risk assessments and the quantitative risk assessment for mammalian wildlife is based on the same data as used in the human health risk assessment. For birds, the most relevant data for this risk assessment are the standard dietary and bird reproduction studies required for registration as well as the acute oral LD_{50} studies. The acute oral LD_{50} values of triclopyr range from 849 mg/kg to 2055 mg/kg, similar to the range seen in experimental mammals. Several subchronic dietary studies have been conducted on triclopyr acid, triclopyr TEA, and triclopyr BEE (Garlon 4). Based on these studies, the U.S. EPA/OPP (1998a) has classified triclopyr acid as being practically non-toxic to slightly toxic to birds and triclopyr TEA and triclopyr BEE (Garlon 4) as practically non-toxic to birds. As in experimental mammals, triclopyr has also been tested for reproductive effects in birds. The LOAEL for reproductive toxicity in birds is 500 ppm in the diet or about 50 mg/kg bw with a corresponding NOAEL of 20 ppm in the diet or about 20 mg/kg bw. These values are marginally higher than the NOAEL of 5 mg/kg bw and LOAEL of 25 mg/kg bw in mammals. Based on standard bioassays in the honey bee, U.S. EPA has classified triclopyr as practically non-toxic to bees. No additional studies on the toxicity of triclopyr or triclopyr formulations to terrestrial invertebrates have been encountered. Little information is available on the toxicity of triclopyr to terrestrial microorganisms. Very high concentrations of triclopyr has been shown to cause growth inhibition in bacteria and fungi in laboratory bioassays.

Triclopyr mimics indole auxin plant growth hormones and cause uncontrolled growth in plants. The U.S. EPA requires studies of seedling emergence and vegetative vigor in non-target plants for herbicides. Triclopyr BEE is about equally toxic in both types of assays with the lowest

NOEC being 0.0036 lb/acre for seeding emergence and 0.0039 lb/acre for vegetative vigor. Triclopyr TEA, on the other hand, is much less toxic in the seedling emergence assay, with a NOEC of 0.333 lb/acre. For the most sensitive species tested, the NOEC for triclopyr TEA in the vegetative vigor assay is 0.0041 lb/acre, about the same as that of triclopyr BEE. The least sensitive species, however, had a much higher NOEC of 0.0111 lb/acre. As field study indicates that some bryophytes and lichens may be sensitive to long term effects after triclopyr exposure.

In addition to the laboratory bioassays and field observations on single species or related groups of species, there are a number field studies that have assessed the effects of triclopyr on terrestrial organisms, both animal and plant. There is very little suggestion in any of the field studies that triclopyr had any direct adverse effect on terrestrial species and most reported effects may simply reflect changes in habitat secondary to vegetation management practices.

As with terrestrial species, the acute lethal potency of triclopyr and triclopyr formulations has been relatively well-defined. There is a major difference in the potential hazards posed by triclopyr TEA formulations (e.g., Garlon 3A) and triclopyr BEE formulations (e.g., Garlon 4) to fish but there are no remarkable differences among species in terms of sensitivity to the various agents covered in this risk assessment. The sublethal effects of Garlon 4 on a salmonid (rainbow trout) has been assayed: at concentrations of 0.32-0.43 mg/L, about a factor of 2 below the 96-hour LC_{50}, fish were lethargic. At levels $\leq$0.1 mg/L, fish were hypersensitive over 4-day periods of exposure. This is reasonably consistent with the threshold for behavioral changes in rainbow trout for Garlon 4 of 0.6 mg/L. The corresponding threshold for behavioral changes to Garlon 3A was 200 mg/L is consistent with the relative acute lethal potencies of these two agents. Subchronic toxicity data are available only on the triethylamine salt of triclopyr and only in fathead minnows. The survival of fathead minnows (embryo-larval stages) was significantly reduced at 253 mg/L compared with control animals. At 162 mg/L, there was a slight decrease in body length.

The observation of hind limb deformities in free-living amphibians has substantially increased concern for the effects of xenobiotics on populations of amphibians. Garlon 3A and Garlon 4 have been specifically tested for malformations in the frog embryo teratogenesis assay and no statistically significant effects were noted. In studies on embryos and tadpoles of three species of frogs using Garlon 4, exposures to 0.6, 1.2, and 4.6 ppm a.e. caused no effect on hatching success, malformations, or subsequent avoidance behavior of embryos but the two higher concentrations were associated with mortality or immobility in tadpoles.

Based on acute lethality, aquatic invertebrates appear to be about equally or somewhat less sensitive than fish to the various forms of triclopyr. The only chronic toxicity data involves a reproduction study in daphids in which the NOEC was 80.7 mg/L with a corresponding LOEC of 149 mg/L.

Based on EC_{50} values, triclopyr TEA is about equally toxic to both algae (lowest EC_{50} of 5.9 ppm a.i.) and macrophytes (lowest EC_{50} of 8.8 ppm a.i.). As with toxicity to fish and invertebrates,

triclopyr BEE is more toxic with EC_{50} values as low as 0.88 ppm a.i. for macrophytes and 0.1 ppm for algae. Efficacy studies are available on the use of Garlon 3A to control unwanted aquatic vegetation. At levels of 0.25-2.5 mg a.e./L (as Garlon 3A) over time periods of 2-48 hours, very little effect was seen for exposure periods less than 6 hours. At 0.25 mg/L, effective control was associated with exposure periods of 24 (partially effective) to 72 (very effective) hours.

TCP (an environmental metabolite of tryclopyr) is substantially more toxic in fish than either triclopyr acid or triclopyr TEA, with acute LC_{50} values in the range of about 2 to 10 ppm, similar to the toxicity of triclopyr BEE. An early life-stage study has been conducted with TCP in rainbow trout yielding an NOEC of 0.0808 mg/L and an LOEC of 0.134 mg/L based on the most sensitive endpoint. Thus, TCP appears to be much more toxic than triclopyr TEA, for which the corresponding values in an early life stage study in the fathead minnow are 104 mg/L and 162 mg/L.

Exposure Assessment – Terrestrial animals might be exposed to any applied herbicide from direct spray, the ingestion of contaminated media (vegetation, prey species, or water), grooming activities, or indirect contact with contaminated vegetation. The highest exposures for terrestrial vertebrates will occur after the consumption of contaminated vegetation or contaminated insects. In acute exposure scenarios, doses as high as 112 mg/kg are estimated. Other routes of exposure, like the consumption of contaminated water or direct spray, lead to lower levels of exposure. In chronic exposure scenarios, the higher estimated daily doses are in the range of about 1 to 29 mg/kg/day and are associated with highly conservative assumptions regarding the consumption of contaminated vegetation.

The primary hazards to non-target terrestrial plants are associated with unintended direct deposition or spray drift. Unintended direct spray will result in an exposure level equivalent to the application rate. At least some plants that are sprayed directly with triclopyr at or near the recommended range of application rates will be damaged. Based on the AgDRIFT model, no more than 0.0058 of the application rate would be expected to drift 100 m offsite after low boom ground applications. In order to encompass a wide range of field conditions, GLEAMS simulations were conducted for clay, loam, and sand at annual rainfall rates from 5 to 250 inches. Under arid conditions (i.e., annual rainfall of about 10 inches or less), there is no or very little runoff. Under these conditions, degradation, not dispersion, accounts for the decrease of triclopyr concentrations in soil. At higher rainfall rates, plausible offsite movement of triclopyr results in runoff losses that range from about negligible up to about 0.4 of the application rate, depending primarily on the amount of rainfall rather than differences in soil type, with somewhat greater runoff predicted for triclopyr TEA compared to triclopyr BEE.

For triclopyr TEA, the potential for effects on aquatic species are based on estimated concentrations of triclopyr in water that are identical to those used in the human health risk assessment without additional elaboration. The maximum concentrations of triclopyr in water from the direct application of Garlon 3A for the control of submerged weeds will be similar to

the lower to central estimates of concentrations of triclopyr in water after an accidental spill of Garlon 3A. An elaboration of the exposure assessment for triclopyr BEE is, however, required because there are substantial differences in the toxicity of triclopyr TEA and triclopyr BEE to aquatic species and substantial differences in the environmental fate of triclopyr TEA and triclopyr BEE. For this risk assessment, a separate set of GLEAMS models were made using triclopyr BEE as the parent compound and triclopyr acid as the metabolite.

Dose-response Assessment – The dose-response assessment for terrestrial mammals is based on the same toxicity values that form the basis of the RfDs used in the human health risk assessment: an acute NOAEL of 100 mg/kg/day and a chronic NOAEL of 5 mg/kg/day. For birds, the acute NOAEL is taken as 535 mg/kg/day for triclopyr acid and 388 mg/kg/day for triclopyr BEE. These based on the 5-day dietary concentrations of 5357 ppm acid equivalents and 3884 ppm acid equivalents for triclopyr TEA and triclopyr BEE. For chronic exposures, the NOAEL is taken as 10 mg/kg/day for both forms of triclopyr. Because triclopyr BEE is rapidly converted to triclopyr acid, chronic exposures to triclopyr BEE are implausible. The only information on the toxicity of triclopyr to terrestrial invertebrates is the standard studies in honey bees that are required for pesticide registration that report an LD_{50} values were over 100 µg/bee. Laboratory studies involving responses in artificial growth media suggest that responses in soil microorganisms may be highly variable among species with growth unaffected in some species at concentrations of up to 1000 ppm in growth medium but inhibited in other species are concentrations as low as 0.1 ppm. The applicability of these studies to assessing the risk to soil microorganisms from exposures to triclopyr in soil is questionable but these are the only data available.

For terrestrial plants, the risk characterization for triclopyr will be based on the standard assays used by U.S. EPA for both triclopyr TEA and triclopyr BEE. For vegetative vigor, the most sensitive NOAEL for triclopyr TEA is 0.0041 lb/acre and the corresponding value for triclopyr BEE is 0.0039 lb/acre and both of these values are used directly for the risk characterization. For triclopyr TEA, the risk characterization for effects on seedling emergence from runoff will be based on the NOAEL of 0.333 lb/acre. The corresponding value for triclopyr BEE will be taken as 0.003 lb/acre.

For aquatic species, the U.S. EPA typically uses LC_{50} values or fractions of LC_{50} values as the basis for characterizing risk of acute exposures in fish. In the U.S. EPA/OPP RED on triclopyr, an acute LC_{50}equivalent to 199 ppm a.e. is used to characterize acute risks to freshwater fish for triclopyr TEA and an acute LC_{50} value of 0.25 ppm a.e. is used to characterize acute risks for freshwater fish for triclopyr BEE. For the quantitative risk characterization, the LC_{50} values selected by U.S. EPA/OPP are maintained in this risk assessment.

Data on subchronic and chronic toxicity to fish is scant. Only one subchronic toxicity is available reporting a NOEC of 104 mg/L for triclopyr TEA. This study is relevant both to triclopyr TEA and triclopyr BEE because of the rapid hydrolysis of triclopyr BEE. Thus, for both triclopyr TEA and triclopyr BEE, the NOAEL of 104 mg/L is used to assess chronic toxicity in

fish. There are relatively few studies available on amphibians. Because fish are apparently more sensitive to triclopyr, both TEA and BEE, and because of the more extensive toxicity data available on fish, a separate dose-response assessment for amphibians is not conducted. Aquatic invertebrates appear to be as sensitive to both triclopyr TEA and triclopyr BEE as are fish. For this risk assessment, an LC_{50} values of 132.9 mg/L for triclopyr TEA and 8.55 mg/L for triclopyr BEE will be use to characterize acute risks to aquatic invertebrates. For chronic effects on invertebrate species, a chronic NOEC of 80.7 mg/L from a daphnid reproduction study is used for risk characterization.

Risk Characterization – For terrestrial mammals, the central estimates of hazard quotients do not exceed the level of concern for any exposure scenarios. At the upper range of exposures, the hazard quotients exceed the level of concern for large mammals and large birds consuming contaminated vegetation exclusively at the application site.

At higher application rates, concern for exposure scenarios involving the consumption of contaminated vegetation is augmented substantially. At the maximum application rate of 10 lbs a.e./acre, the central estimate of the hazard quotient exceed the level of concern for several acute exposure scenarios: the direct spray of a small mammal assuming 100% absorption, a large mammal consuming contaminated vegetation, and a small bird consuming contaminated insects. The central estimates of the hazard quotients for the chronic consumption of vegetation is exceeded for a large mammal and a large bird and the upper range on the hazard quotients are also increased by a factor of 10: i.e., to 60 for a large mammal and 50 for a large bird. This risk assessment is consistent with the risk characterization given by U.S. EPA indicating that contaminated vegetation is primary concern in the used of triclopyr and that high application rates will exceed the level of concern for both birds and mammals in longer term exposure scenarios.

Some effects may be anticipated on nontarget vegetation under some conditions. Because of the relatively low toxicity of triclopyr TEA compared to triclopyr BEE, the risk characterization for triclopyr TEA is much less severe than that of triclopyr BEE. At an application rate of 1 lb/acre, potentially damaging runoff from triclopyr TEA would be anticipated only at relatively high rainfall rates. While a lesser amount of triclopyr BEE will runoff, the higher toxicity of triclopyr BEE leads to hazard quotients above the level of concern starting are relatively modest rainfall rates – i.e., 20 to 25 inches per year. At an application rate of 10 lbs a.e./acre per acre, damage due to runoff after the application of triclopyr TEA would be expected at annual rainfall rates as low as 20 inches per year. For triclopyr BEE, the hazard quotients are of concern for all but the most arid areas. The potential impact of offsite drift of triclopyr varies substantially with the application rate. At an application rate of 1 lb a.e./acre, potentially damaging exposures could occur within about 100 feet of the application site. At the maximum application rate of 10 lbs a.e./acre, damaging drift could occur at distances of over 1000 feet from the application site.

The risk characterization for aquatic organisms differs for triclopyr TEA and triclopyr BEE. For triclopyr TEA, risks to aquatic species are low over the entire range of application rates that may

be used in Forest Service programs. At the highest projected application rate, the hazard quotient for acute risks to aquatic plants from runoff into streams would reach unity. For acute risks to aquatic plants in the application of triclopyr TEA directly to water for the control of submerged weeds, the hazard quotient of 0.6 is based on the targeted water concentration given on the product label.

Although triclopyr BEE is much more toxic to aquatic species than triclopyr TEA or triclopyr acid, the projected levels of exposure are much less even for acute scenarios because of the rapid hydrolysis of triclopyr BEE to triclopyr acid as well as the lesser runoff of triclopyr BEE because of it's lower water solubility and higher affinity for soils. Nonetheless, triclopyr BEE is projected to be somewhat more hazardous when used near bodies of water where runoff to open water may occur. At an application rate of 1 lb a.e./acre, the level of concern for acute exposure to aquatic plants is exceeded at the upper range of projected concentrations. At an application rate of 10 lbs a.e./acre, the level of concern for acute exposure to aquatic plants is exceeded at the central estimate as well as the upper range of projected concentrations.

The risk characterization for TCP is considered quantitatively only for fish because toxicity data are available only for fish. At the typical application rate of 1 lb a.e./acre, the worst case hazard quotients are below the level of concern. That the maximum application rate of 10 lbs a.e./acre, the hazard quotients would be a factor of 10 higher and the hazard quotient for longer term exposure would be substantial (HQ=9). Thus, if triclopyr is applied at higher rates of exposure in areas where surface water contamination is plausible, site-specific modeling and/or environmental monitoring would be useful to ensure and verify that concentrations TCP do reach harmful concentrations. Concentrations of TCP in surface water after the application of triclopyr at 1 lb a.e./acre and chlorpyrifos at 1 lb a.e./acre are well below a level of concern. Thus, the concern for TCP residues in surface water appears to be associated with high application rates of triclopyr rather than applications triclopyr and chlorpyrifos in the same area.

1. INTRODUCTION

This document provides risk assessments for human health effects and ecological effects to support an assessment of the environmental consequences of using triclopyr in Forest Service vegetation management programs. This risk assessment is an update to the previous USDA Forest Service risk assessment of triclopyr (SERA 1996). Five commercial formulations of triclopyr are currently registered for forestry applications and are covered in this risk assessment. One of these, Garlon 3A, contains the triethylamine (TEA) salt of triclopyr. The other four formulations (Garlon 4, Forestry Garlon 4, Pathfinder II, and Remedy RTU) contain the butoxyethyl ester (BEE) of triclopyr. An additional formulation of the triethylamine (TEA) salt of triclopyr, Renovate 3, has been labeled for aquatic weed control and this use is also considered in the current risk assessment.

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

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

In the preparation of this risk assessment, literature searches of triclopyr were conducted in the open literature using PubMed, TOXLINE as well as the U.S. EPA CBI files. In addition to these standard literature searches, additional sources of information were used including the U.S. EPA Reregistration Eligibility Decision document on triclopyr (U.S. EPA/OPP 1998a), the EXTOXNET review of this compound (Extoxnet 1996), various reviews submitted to U.S. EPA to support the registration of triclopyr (Eisenbrandt et al. 1997; Houtman and Mayes 1997; Houtman et al. 1997c; Knuteson 1999; McMaster 1997; Wolt 1997; Wolt et al. 1997), and a review of environmental concerns with the use of triclopyr (Cox 2000).

The search of U.S. EPA's FIFRA/CBI files indicated that there is a complete set of standard studies conducted for this compound - i.e., a total of over 1117 submissions. Many of these studies were conducted to support the initial registration and reregistration of triclopyr, a substantial number of studies were conducted and submitted to U.S. EPA prior to 1998, the date of the U.S. EPA Reregistration Eligibility Decision document on triclopyr (U.S. EPA/OPP

1998a). Relatively few studies were submitted to U.S. EPA after the publication of the RED and most of these studies involve formulations that do not involve forestry applications and are not used by the Forest Service. The reregistration document for triclopyr (U.S. EPA/OPP 1998a) was used where possible to summarize information for the earlier CBI studies. Although full copies of some key studies were obtained from the earlier literature, the acquisition of the CBI studies focused on the post-1998 period. Full text copies of the most relevant CBI studies [n=142] were kindly provided by the U.S. EPA Office of Pesticide Programs. These include all key studies cited in the RED as well as some newer studies on effects of major concern (e.g., the egg and fry study in trout by Marino et al. (1999). The CBI studies were reviewed, and synopses of the most relevant studies are included in the appendices to this document. Those studies summarized from the RED (U.S. EPA/OPP 1998a) are cited simply by MRID number. Studies that were obtained and reviewed are indicated by the standard author-date citation followed by the MRID number.

The human health and ecological risk assessments presented in this document are not, and are not intended to be, comprehensive summaries of all of the available information. The information presented in the appendices and the discussions in chapters 2, 3, and 4 of the risk assessment are intended to be detailed enough to support a review of the risk analyses; however, they are not intended to be as detailed as the information generally presented in Chemical Background documents or other comprehensive reviews. To review each study would far exceed the resources available to the Forest Service and, more importantly, would make the document very difficult to read and review. In some respects, an all inclusive and detailed review of each study would tend to obscure rather than inform the risk assessment.

As an alternative, this document focuses on information that is likely to impact the risk assessments. This information was identified from a screening of each of the identified citations using available abstracts, key words, and other available details. In addition, the relevance of studies was also assessed by consulting the available reviews, detailed above. Nonetheless, the selection of studies for inclusion into this risk assessment is an admittedly judgmental process. In order to maintain transparency, this risk assessment is accompanied by a complete bibliography of all studies encountered in the literature search. This bibliography is included as Attachment 1 and indicates which documents were retrieved.

The Forest Service will update this and other similar risk assessments on a periodic basis and welcomes input from the general public on the selection of studies included in the risk assessment. This input is helpful, however, only if recommendations for including additional studies in the body of these risk assessments specify why and/or how the new or not previously included information would be likely to alter the conclusions reached in the risk assessments.

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

Risk assessments are usually expressed with numbers; however, the numbers are far from exact. *Variability* and *uncertainty* may be dominant factors in any risk assessment, and these factors should be expressed. Within the context of a risk assessment, the terms *variability* and *uncertainty* signify different conditions.

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

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

In considering different forms of variability, almost no risk estimate presented in this document is given as a single number. Usually, risk is expressed as a central estimate and a range, which is sometimes very large. Because of the need to encompass many different types of exposure as well as the need to express the uncertainties in the assessment, this risk assessment involves numerous calculations.

Most of the calculations are relatively simple, and the very simple calculations are included in the body of the document. Some of the calculations, however, are cumbersome. For those calculations, a set of worksheets is included with the risk assessment. The worksheets provide

the detail for the estimates cited in the body of the document. The worksheets are divided into the following sections: general data and assumptions, chemical specific data and assumptions, exposure assessments for workers, exposure assessments for the general public, and exposure assessments for effects on nontarget organisms. Detailed documentation for these worksheets are included as Attachment 1.

Because the properties of triclopyr acid and triclopyr BEE result in substantially different exposure estimates for a few scenarios, two sets of worksheets, Supplement 1 and 2, are provided with this risk assessment covering triclopyr acid and triclopyr BEE. Two versions of each set of worksheets are available: one in a word processing format and one in a spreadsheet format. The worksheets that are in the spreadsheet format are used only as a check of the worksheets that are in the word processing format. Both sets of worksheets are provided with this risk assessment. Documentation for the use of these worksheets is provided as Attachment 2.

2. PROGRAM DESCRIPTION

2.1. OVERVIEW

Triclopyr is a herbicide that mimics auxin, a plant growth hormone, thus disrupting the normal growth and viability of plants. Triclopyr is used in Forest Service programs primarily for wildlife habitat improvement, noxious weed control, conifer or hardwood release, and site preparation, with other minor uses including rights-of-way management, hardwood control, facilities maintenance, and seed orchard protection. Two forms of triclopyr are used commercially as herbicides: the triethylamine salt and the butoxyethyl ester. Currently, there are 5 commercial formulations of triclopyr that are registered for forestry applications: Garlon 3A, Garlon 4, Forestry Garlon 4, Pathfinder II, and Remedy RTU. Garlon 3A contains the triethylamine salt of the triclopyr and inert ingredients and requires the use of a non-ionic surfactant. In addition to triethylamine, Garlon 3A contains EDTA, a common chelating agent, and ethanol. Garlon 3A, marketed as Renovate 3, has recently been labeled for aquatic weed control. The other commercial formulations contain the butoxyethyl ester of triclopyr, often referred to as triclopyr-BEE. Garlon 4 and Forestry Garlon 4 both contain kerosene and proprietary surfactants. For this risk assessment and based on recent Forest Service applications, the average application rate is taken as 1 lb a.e./acre with a range of 0.05 lb a.e./acre to 10 lbs a.e./acre. The total annual use of triclopyr by the Forest Service for 2001 is about 7,700 lbs, which is about 4 percent of the agricultural use. Nonetheless, the agricultural uses of triclopyr are rather localized and, in some regions, the use of triclopyr in Forest Service programs may be a substantial source of triclopyr residues in the environment.

2.2. CHEMICAL DESCRIPTION AND COMMERCIAL FORMULATIONS

Triclopyr is the common name for [(3,5,6-trichloro-2-pyridinly)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 2-1). Like 2,4,5-T, triclopyr mimics auxin, a plant growth hormone, thus disrupting the normal growth and viability of plants.

Two forms of triclopyr are used commercially as herbicides: the triethylamine salt and the butoxyethyl ester. The structure of both of these forms is also illustrated in Figure 2-1. Some basic chemical and physical properties of triclopyr and triclopyr BEE are summarized in Table 2-1 and Table 2-2, respectively. At ambient temperatures, triclopyr is a fluffy solid (Budavari et al. 1989) and is readily soluble in water (Table 2-1). In aqueous solutions, the hydrogen atom of the carboxylic acid group (**COOH**) may be associated (e.g., **-COOH**) or dissociated (e.g., $\mathbf{-COO^-}$ + $\mathbf{H^+}$) depending on the pH of the solution. The dissociation constant, or pK_a, for the carboxylic acid group is approximately 3. Thus, at a pH of 3, 50% of the acid is associated and 50% is disassociated. As the acidity of the solution decreases (i.e., the pH of the solution increases) the proportion of triclopyr that is ionized or dissociated increases. The pH of most biological fluids ranges from approximately 5 to 9. Thus, within this range of pH, most of the triclopyr acid has a net negative charge ($\mathbf{-COO^-}$).

As discussed in Section 2.4, application rates for triclopyr are expressed in this risk assessment in units of acid equivalents (a.e.) rather than active ingredients (a.i.). For triclopyr, active ingredients refers to the TEA salt or BEE ester. Many of the toxicity studies conducted on triclopyr, summarized in the appendices to this risk assessment, report exposures in units of a.i. rather than a.e. For the risk characterization, concentrations or doses in units of a.i. are converted to units of a.e. by multiplying the a.i. value by the ratio of the molecular weight of triclopyr acid (256.48 g/mole) to the molecular weight of the a.i. – 371.7 g/mole for triclopyr TEA or 356.64 g/mole for triclopyr BEE. Thus, the conversion factors are 0.690 for triclopyr TEA [256.48/371.7] and 0.719 for triclopyr BEE [256.48/356.64].

The previous USDA Forest Service risk assessment of triclopyr (SERA 1996) covered only two formulations: Garlon 3A and Garlon 4. Currently, there are 5 commercial formulations of triclopyr that are registered for forestry applications: Garlon 3A, Garlon 4, Forestry Garlon 4, Pathfinder II, and Remedy RTU. An additional formulation for the control of aquatic vegetation, Renovate 3, is available from SePRO Corporation (SePRO 2003a and b) and appears to be identical to Garlon 3A. Information on each of these formulations are detailed in Appendix 1. Garlon 3A contains the triethylamine salt of the triclopyr (44.4%) and inert ingredients (55.6%) and requires the use of a non-ionic surfactant. In addition to triethylamine, Garlon 3A contains EDTA, a common chelating agent, and ethanol (≈ 1%). The other commercial formulations contain the butoxyethyl ester of triclopyr, often referred to as triclopyr-BEE. Garlon 4 and Forestry Garlon 4 both contain 61.6% triclopyr-BEE and 38.4% inerts. For both formulations, the inerts include kerosene and proprietary surfactants. Additional surfactants or oils are recommended for both products for some types of applications. Pathfinder II and Remedy RTU are both "ready to use" formulations – i.e., require no mixing and no addition of surfactants or other adjuvants – and both contain 13.6% triclopyr-BEE and 86.4% inert ingredients. The inert ingredients in these formulations are specified only as "proprietary surfactants" in the open literature (C&P Press 2002).

Information on the amount of kerosene in Garlon 4 is not available in the open literature. The formulation must contain at least 1% of the inert to require that the inert be identified on the label. This may be taken as the lower limit of the concentration of kerosene in Garlon 4. As summarized in (SERA 1996), DowElanco indicated that no individual inert is present at greater than 6% in Garlon 3A or Garlon 4.

Inerts are classified by the U.S. EPA as ranging from inerts of toxicologic concern (List 1) to inerts of minimal concern (List 4) (U.S. EPA/OPP 2001a). Some inerts - i.e., those listed under SARA Title III, Section 313 - are specified on the product material safety data sheets, as specified in Appendix 1, and can be publicly disclosed. The specific identity of the surfactants and other inerts has been disclosed to the U.S. EPA as part of the registration process. As part of the current risk assessment, information on the inerts in the commercial formulations of triclopyr has been obtained and reviewed (e.g., Hill 1999a,b; Hill 2000, Hill 2001). The specific identity of these inerts, however, cannot be disclosed in this risk assessment.

2.3. APPLICATION METHODS

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

Hack and squirt applications are a form of cut surface treatment in which the bark of a standing tree is cut with a hatchet and the herbicide is applied with a squirt bottle. This treatment method is used to eliminate large trees during site preparation, conifer release operations, or rights-of-way maintenance. As with selective foliar applications, a worker usually treats about 0.5 acres/hour with a plausible range of 0.25-1.0 acres/hour.

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

Boom spray or roadside hydraulic spraying is used primarily in rights-of-way management. Spray equipment mounted on tractors or trucks is used to apply the herbicide on either side of the roadway. Usually, about 8 acres are treated in a 45-minute period (approximately 11 acres/hour) with approximately 200 gallons of the herbicide mixture (270 gallons/hour). Some special truck mounted spray systems may be used to treat up to 12 acres in a 35-minute period with approximately 300 gallons of herbicide mixture (approximately 21 acres/hour and 510 gallons/hour) (USDA 1989b, p 2-9 to 2-10).

Aerial applications are restricted to the use of helicopters (i.e., fixed wing aircraft may not be used). Liquid formulations of triclopyr are applied under pressure through specially designed spray nozzles and booms. The nozzles are designed to minimize turbulence and maintain a large droplet size, both of which contribute to a reduction in spray drift. In aerial applications, approximately 40-100 acres may be treated per hour.

In some instances, areas treated with triclopyr may be subject to brown-and-burn operations. As discussed in USDA (1989b), these operations involve burning a treated area 30–180 days after treatment with the herbicide.

DowElanco, now referred to as Dow AgroSciences, was granted an experimental use permit for the application of triclopyr to control unwanted aquatic vegetation (U.S. EPA/OPP 1997) and Garlon 3A has recently been labeled for aquatic weed control (Dow AgroSciences 2002). This use has only been permitted recently and the direct application of Garlon 3A to standing water for the control of submerged aquatic weeds has not been a part of Forest Service program activities. The distribution and sales of triclopyr TEA for this application appears to have been recently transferred or licenced to SePRO Corporation (SePRO 2003a and b), which markets the product as Renovate 3. Based on the product label, Renovate 3 may be applied at rates of 2 qt/acre to 8 qt/acre, equivalent to 1.5 to 6 lb/acre and target concentrations in water for the control of submerged weeds range from 0.75 to 2.5 ppm. Water concentrations near potable water intakes must be less than 0.4 mg/L. The Forest Service may consider the use of Renovate 3 to control aquatic vegetation and this use is considered in the human health (Section 3.2.3.4.4) and ecological risk assessments (4.2.4).

2.4. USES AND APPLICATION RATES

The use of triclopyr in Forest Service Programs for fiscal year 2001, the most recent year for which data are available, is detailed in Appendix 2 (use by region) and Appendix 3 (use by program activity). Triclopyr is used in a variety of Forest Service programs (Table 2-3). The greatest use, in terms of the amount applied, involves wildlife habitat improvement (about 25%) but substantial use also occurs in noxious weed control, conifer or hardwood release, and site preparation (about 20% each). The remainder of the triclopyr used by the Forest Service involves rights-of-way management (about 10%) with marginal (<1%) use in hardwood control, facilities maintenance, and seed orchard protection. Average application rates for most uses range from about 1 to 1.5 lbs/acre for most applications, although lesser application rates (about 0.4 to 0.7 lbs/acre) are used for release, hardwood control, and site preparation.

For this risk assessment, the average application rate will be taken as 1 lb a.e./acre. This is an essentially arbitrary selection but is reasonably close to application rates used in several types of program activities (Table 2-3). The highest recorded application rate for 2001 is 10.29 lbs/acre. As specified in Appendix 2, this application was used by Region 8 in right-of-way vegetation management. This is near the application rate of 9.88 lbs/acre used by Region 2 in noxious weed control. All of these application rates are in units acid equivalents (a.e.) per acre.

For the current risk assessment, the highest application rate used for the risk assessment with be 10 lbs a.e./acre. This should encompass the highest application rate that could be anticipated in Forest Service applications. An application rate of 0.1 lb a.e./acre will be used as the lower range of the application rate. This is somewhat arbitrarily selected. As detailed in Appendix 3, the lowest application rate used by the Forest Service in 2001 is 0.05 lb a.e./acre.

For this risk assessment, the extent to which a formulation of triclopyr is diluted prior to application primarily influences dermal and direct spray scenarios, both of which are dependent on ‘field dilution’(i.e., the concentration of triclopyr in the applied spray). In all cases, the higher

the concentration of triclopyr - equivalent to the lower dilution of the triclopyr formulation - the greater the risk.

The lowest dilution recommended for ground or aerial applications of Garlon 4 is 5 gallons per acre (Appendix 1) and this is appropriate for Forest Service programs. The highest dilution recommended for ground applications of Garlon 3A or Garlon 4 is on 100 gallons of water per acre. For Forest Service programs, however, this high of a dilution would be atypical and the upper range on the dilution volume used in this risk assessment is 40 gallons per acre. A typical dilution rate is taken as 25 gallons per acre. Details regarding the calculation of field dilution rates are given in worksheet B01, and the calculations following this worksheet are summarized in worksheet B02. It should be noted that the selection of application rates and dilution volumes in this risk assessment is intended to simply reflect typical central estimates as well as plausible lower and upper ranges. In the assessment of specific program activities, the Forest Service will use program specific application rates in the worksheets that are included with this report to assess any potential risks.

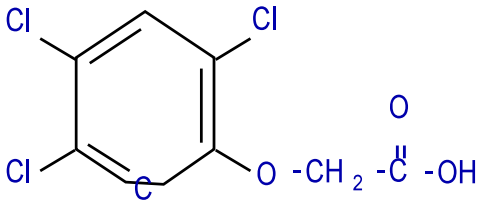
2.5. USE STATISTICS

The USDA Forest Service (USDA/FS 2002) tracks and reports use by geographical areas referred to as "*Regions*". As illustrated in Figure 2-2, the Forest Service classification divides the U.S. into nine regions designated from Region 1 (Northern) to Region 10 (Alaska). [Note: There is no *Region 7* in the Forest Service system.] As illustrated in Figure 2-2 and detailed further in Table 2-4, by far the greatest use of triclopyr occurs in the southeast, referred to by the Forest Service as Regions 8 or the southern region. This region used about 80% of the triclopyr used by the Forest Service in 2001. Relatively small amounts (about 5% each) are used in Region 2 (Rocky Mountains), Region 6 (Pacific Northwest), and Region 9 (Eastern). In other regions, the use of triclopyr is insubstantial (about 1% or less of total Forest Service use)

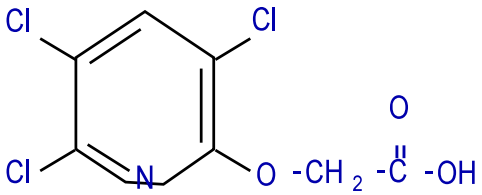
Triclopyr formulations are used in agriculture. As illustrated in Figure 2-3, about 200,000 lbs [192,606 lbs] of triclopyr are applied to crops annually, primarily to pastures and rice. These use statistics are for 1992, the most recent year for which data are available (USGS 1998). As noted in Table 2-3, the total annual use of triclopyr by the Forest Service for 2001 is about 7,700 lbs, which is about 4 percent of the agricultural use [7,671.69 lbs ÷ 192,606 lbs = 0.0398]. Thus, while the use of triclopyr by the Forest Service is not trivial, this use is less than that of agricultural uses by a factor of about 25. Thus, there is no basis for asserting that Forest Service programs are a substantial source of triclopyr in the environment in terms of total source contribution.

Nonetheless, as also indicated in Figure 2-3, the agricultural uses of triclopyr are rather localized, with most uses occurring in Oregon, Wisconsin, Virginia, West Virginia, Arkansas, and Louisiana. Thus, in some regions, particularly in the southeast, the use of triclopyr in Forest Service programs may be a substantial source of triclopyr residues in the environment. This potential for local contamination of environmental media by the use of triclopyr in Forest Service

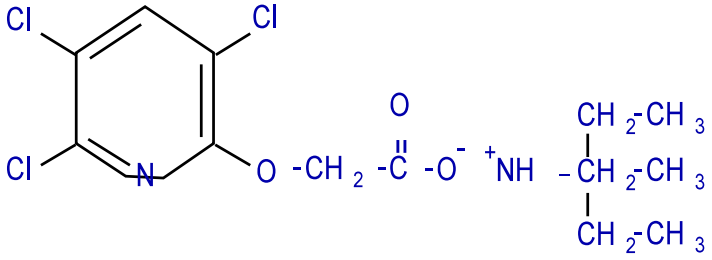
programs is discussed further in the human health risk assessment (Section 3) and the ecological risk assessment (Section 4).



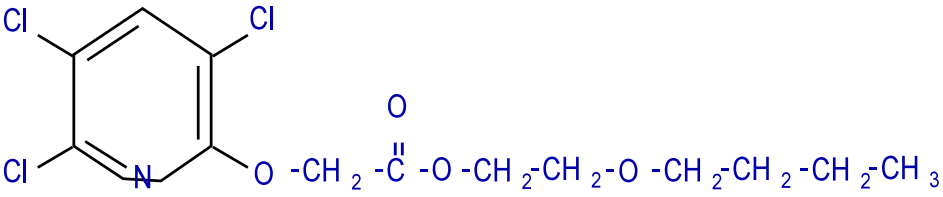
2,4,5-T



Triclopyr acid



Triclopyr, triethylamine salt



Triclopyr, Butoxyethyl ester (TBEE)

Figure 2-1: Structure of the Various Forms of Triclopyr and 2,4,5-T.

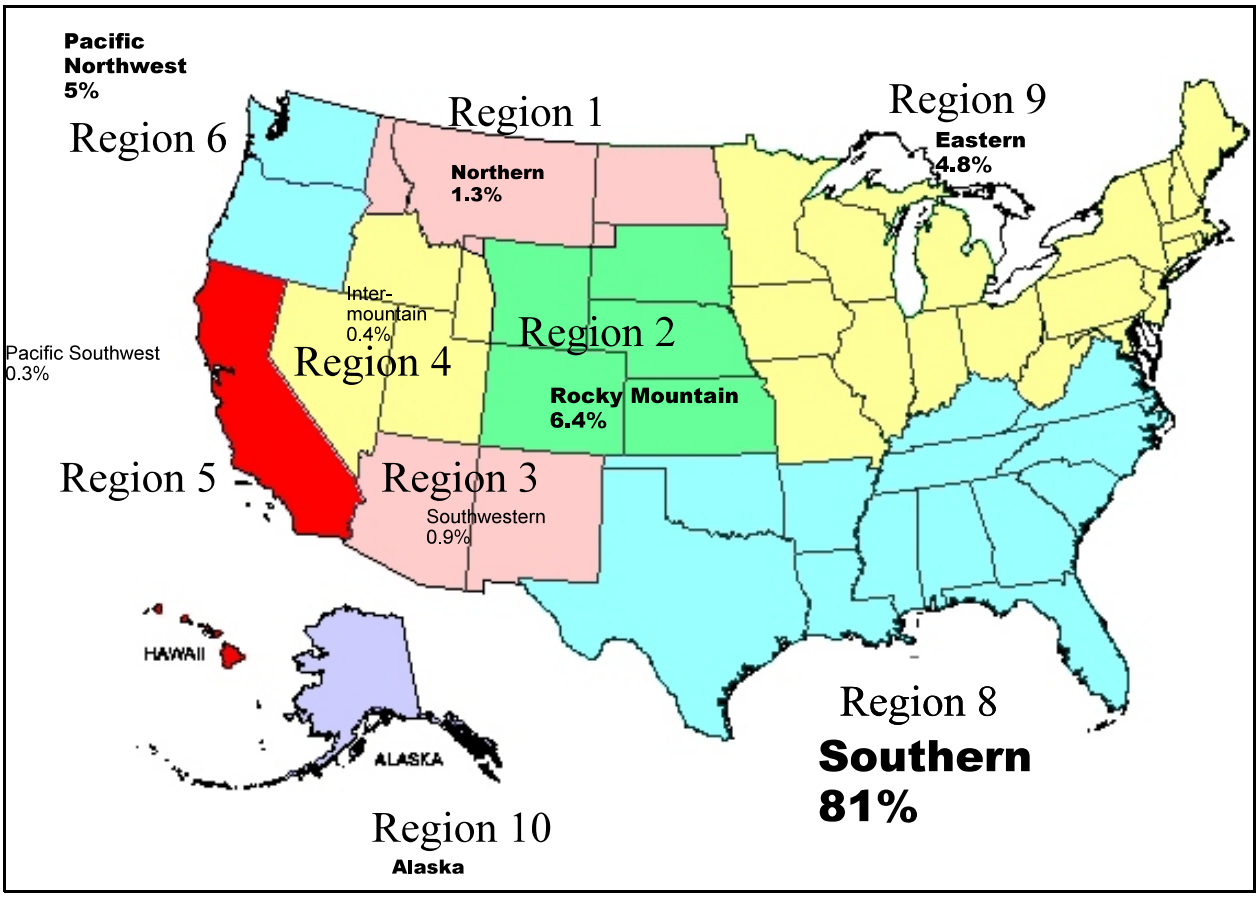


Figure 2-2: Use of triclopyr by the USDA Forest Service in various regions of the United States.

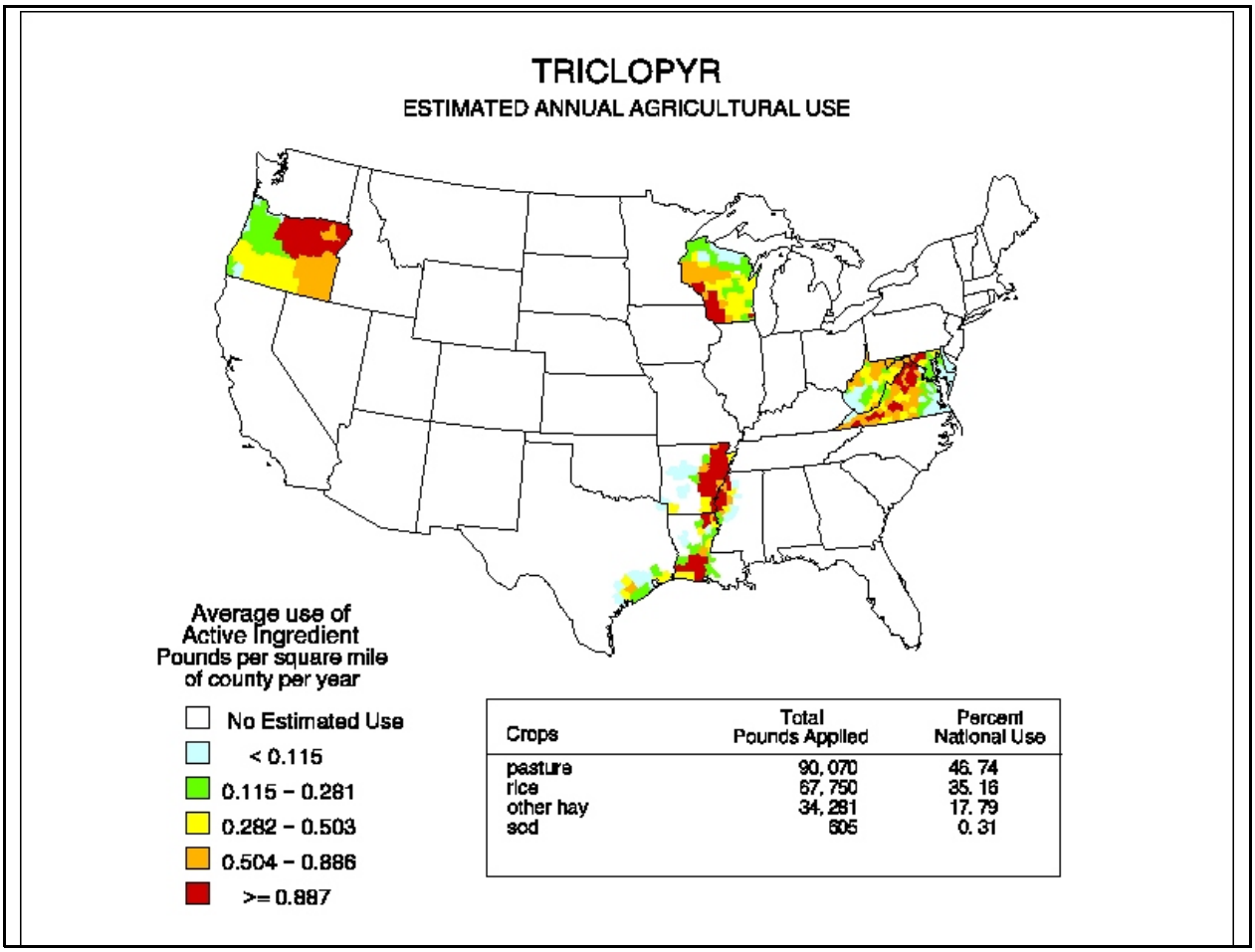


Figure 2-3: Agricultural use of triclopyr in the United States for 1992 (USGS 1998).

Table 2-1. Physical, chemical, and biochemical properties of triclopyr.

CAS Number:	55335-06-3 (USDA/ARS 1995)
Molecular Weight:	256.48 (acid, USDA/ARS 1995)
	371.7 (TEA, U.S. EPA/OPP 1998a)
Air Halftime (days):	3.3 (estimated; Meylan and Howard 1993)
Boiling point (◦C):	290 (USDA/ARS 1995)
Density (g/cm^3):	1.85 at 21°C (Tomlin 1994)
Evaporation Rate:	low (Neary et al. 1993)
Foliar Halftime (days):	3-10 (DT50 in plants) (Tomlin 1994)
	4 (metabolism by aquatic plants) (Woodburn et al. 1993b).
	average 42% decline over 6 days of triclopyr applied to various forest vegetation in northern Idaho (Whisenant and McArthur 1989).
Henry's law constant:	9.89 x 10^{-10} atm-m^3/mole (25◦C) (calculated from vapor pressure and water solubility)
Log K_{ow}:	0.42 (pH 5) (Tomlin 1994)
	-0.45 (pH 7) (Tomlin 1994)
	-0.96 (pH 9) (Tomlin 1994)
	2.53 (non-ionized; estimated)(Meylan and Howard 1995)
Melting point (◦C):	150.5 (Tomlin 1994)
	148 -150 (USDA/ARS 1995)
pKa:	3.97 (Tomlin 1994)
	2.7 (McCall and Gavit 1986)
	2.93 (Woodburn et al. 1993a)
	2.68 (Weber 1994)
	2.93 (USDA/ARS 1995)
Soil Adsorption K_{oc}:	59 (Tomlin 1994)
	27 (McCall and Gavit 1986, Kenaga 1980)
	20 (Knisel et al. 1992; Diaz-Diaz and Loague 2001)
Soil Halftime (days):	46 (average) (Tomlin 1994, Weber 1994)
	45 (average) (Neary et al. 1993)
	40 (average) (McCall and Gavit 1986)
	14 (in selected Canadian forest soils) (Stephenson et al. 1990)
Water Halftime:	2.8-14.1 hours (photodegradation in sunlit water; 0-1 m deep) (McCall and Gavit 1986)
	0.71-1.86 days (photodegradation in natural river water) (Woodburn et al. 1993a).
	0.5-3.6 days (field study in Lake Seminole, GA under midsummer conditions) (Woodburn et al. 1993b).
	3.8-4.3 days (field test in northern Ontario) (Solomon et al. 1988).
Photolysis (days^{-1}):	0.00034 [soil] (USDA/ARS 1995)
	2.0 [water] (USDA/ARS 1995)
Vapor Pressure (mm Hg):	1.50 x 10^{-6} mm Hg (25°C) (Tomlin 1994)
	0.168 mPa (USDA/ARS 1995)
Water Solubility:	7690 g/L at pH 5 (20°C) (Tomlin 1994)
	8100 g/L at pH 7 (20°C) (Tomlin 1994)
	8220 g/L at pH 9 (20°C) (Tomlin 1994)
	430 mg/L (25°C) (Neary et al. 1993)
	435 mg/L (25°C) (USDA/ARS 1995)
	2,100 g/L (TEA salt) (Diaz-Diaz and Loague 2001; USDA/ARS 1995)

Table 2-2. Physical, chemical, and biochemical properties of triclopyr butoxyethyl ester.

CAS Number:	64470-88-8
Molecular Weight:	356.64
Melting Point (°C):	-----
Density (g/cm^3):	-----
Vapor Pressure (mm Hg):	-----
Water Solubility:	2.1 mg/L at 25°C (estimated; Meylan and Howard 1994) 6.8 mg/L (U.S. EPA/OPP 1998a) 23.0 mg/L (Knisel and Davis 1999)
Henry's law constant:	5.98×10^{-8} atm-m^3/mole (25°C) (estimated; Meylan and Howard 1991)
Log K_{ow}:	4.01 (estimated; Meylan and Howard 1995)
Soil Adsorption K_{oc}:	560 (estimated; Meylan and Howard 1992) 780 (Knisel et al. 1999)
Evaporation Rate:	-----
Foliar Halftime (days):	15 (Knisel and Davis 1999) average 42% decline over 6 days of triclopyr, butoxyethyl ester applied to various forest vegetation in northern Idaho (Whisenant and McArthur 1989). initial halftime of approximately 10-15 days after aerial application to litter in bush fields of southwest Oregon (Newton et al. 1990).
Soil Halftime (days):	40 (average) (McCall and Gavit 1986) 14 (in selected Canadian forest soils) (Stephenson et al. 1990) 0.125 (degradation to acid) (U.S. EPA/OPP 1998a)
Water Halftime:	12.5-83.4 hours (photodegradation in sunlit water; depth of 0-1 m) (McCall and Gavit 1986) 0.30 days (hydrolysis at 25°C and pH 9; McCall et al. 1988). 8.7 days (hydrolysis at 25°C and pH 7; McCall et al. 1988). 84.0 days (hydrolysis at 25°C and pH 5; McCall et al. 1988). 3.8 - 4.3 days (field test in northern Ontario) (Solomon et al. 1988). 0.5 (degradation to acid) (U.S. EPA/OPP 1998a)
Air Halftime (days):	0.63 (estimated for gas-phase triclopyr butoxyethyl ester) (method of Meylan and Howard 1993)

Table2-3: Use of triclopyr by USDA Forest Service in 2001 by Type of Use (USDA/FS 2002).

Use Classification	Acres	Pounds	Pounds per acre	Proportion of Use by Acres	Proportion of Use by Pounds
Conifer or Hardwood Release	3488.00	1369.25	0.39	0.337	0.178
Hardwood control	81.00	60.00	0.74	0.008	0.008
Housekeeping/Facilities Maintenance	1.20	1.70	1.42	1.16e-04	2.22e-04
Noxious Weed Control	1180.88	1556.07	1.32	0.114	0.203
Right-of-Way Vegetation Management	745.85	1012.42	1.36	0.072	0.132
Seed Orchard Protection	1.00	1.00	1.00	9.65e-05	1.30e-04
Site preparation	2737.00	1624.10	0.59	0.264	0.212
Wildlife Habitat Improvement	2124.00	2047.15	0.96	0.205	0.267
Grand Total	10,358.93	7,671.69	0.74	1	1

Table 2-4: Use of triclopyr by USDA Forest Service in 2001 by Region (USDA/FS 2002)

Forest	Acres	Pounds	lbs/acre	Proportion of Total Acres	Proportion of Total Lbs.
Northern (R1)	157.30	100.52	0.64	0.015	0.013
Rocky Mountain (R2)	103.66	491.22	4.74	0.010	0.064
Southwestern (R3)	132.00	68.00	0.52	0.013	0.009
Intermountain (R4)	167.95	16.40	0.10	0.016	0.002
Pacific Southwest (R5)	85.00	26.40	0.31	0.008	0.003
Pacific Northwest (R6)	327.47	381.05	1.16	0.032	0.050
Southern (R8)	9,269.25	6,220.10	0.67	0.895	0.811
Eastern (R9)	116.30	368.02	3.16	0.011	0.048
Total	10,358.93	7,671.71	0.74	1.000	1.000

3. HUMAN HEALTH RISK ASSESSMENT

3.1. HAZARD IDENTIFICATION

3.1.1. Overview. Studies regarding histopathology and clinical chemistry data on triclopyr suggest that the liver and kidney are the primary target organs. Like most weak acids, triclopyr is excreted primarily in the kidney by an active transport process. The dermal absorption of triclopyr BEE has been measured *in vitro* using flow-through diffusion cells with skin from rats and humans. In addition, an *in vivo* pharmacokinetics study involving oral and dermal exposure to triclopyr is available using human volunteers. Like any chemical, triclopyr at sufficiently high exposure levels can cause toxic effects, including death. Nonetheless, triclopyr has a low order of acute lethal potency. As large number of subchronic and chronic toxicity studies are available on triclopyr. All studies were submitted to U.S. EPA/OPP to the support the registration of triclopyr. Full copies of all studies were obtained and reviewed as part of the current risk assessment. There is no information suggesting that triclopyr causes direct adverse effects on the nervous system, endocrine system, or immune function. At doses which do not cause maternal toxicity, there is not apparent concern for either reproductive or teratogenic effects. At substantially higher doses that are maternally toxic, triclopyr has been shown to result in birth defects. Most of abnormalities have been indicative of delayed growth and have been associated with maternal toxicity. Standard bioassays for carcinogenicity have been 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 – 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 has reviewed these studies and determined that the evidence for carcinogenicity is marginal and has not recommended as 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. TCP is of concern to this risk assessment both because it is a metabolite of triclopyr and because the aggregate risks of exposure to TCP from the breakdown of both triclopyr and chlorpyrifos must be considered. While there is no indication that the general exposures to TCP from the use of triclopyr and chlorpyrifos will result in harmful levels of exposure, this risk assessment does specifically include a consideration of such exposures that may result from specific program activities in the use of triclopyr and chlorpyrifos in forestry applications.

3.1.2. Mechanisms of Action. Although the toxicity of triclopyr to mammals is relatively well characterized (as detailed in subsequent sections) and the mechanism of action in plants is understood, the mechanisms of action in mammals is unclear. In both acute and chronic studies,

as detailed in Section 3.1.5, the primary target organ appears to be the kidney. Since triclopyr is excreted by the kidney and active transport processes are present in the mammalian kidney for triclopyr as well as a large number of other weak acids, the apparent sensitivity of the kidney to triclopyr may be related to relatively high tissue concentrations of triclopyr in the kidney.

Triclopyr is the pyridine analogue of 2,4,5-T. Like 2,4,5-T, the toxicity of triclopyr to plants appears to involve the mimicking of auxin growth hormones (Section 4.1). The mammalian toxicity of 2,4,5-T, particularly the induction of reproductive effects and the toxic effects of 2,4,5-T in humans, is related to the contamination of 2,4,5-T with TCDD (2,3,7,8-tetrachlorodibenzo-p-dioxin) which is formed as an impurity in the synthesis of 2,4,5-T from the chlorination of phenols. Because triclopyr is based on a pyridine ring rather than an aromatic ring, the occurrence of TCDD in triclopyr is not plausible.

3.1.3. Pharmacokinetics and Metabolism. After oral administration of 3 or 60 mg/kg of ^{14}C-triclopyr acid to rats, approximately 89-95% of the dose was recovered in the urine as unmetabolized triclopyr, indicating that at least this proportion of the administered dose was absorbed. Very little residue was recovered in the feces or carcass. Triclopyr is absorbed and excreted relatively rapidly, with half-times for oral absorption and urinary excretion of 3.61 hours and 1.1 hours, respectively. Virtually all of the ingested dose of triclopyr is excreted unchanged in the urine, although 4 minor metabolites are formed (Timchalk et al. 1990). The rapid urinary elimination of triclopyr has also been noted in cattle after oral exposure to triclopyr, with 86.4% of the administered dose eliminated unchanged in the urine and no residues detected in the milk or feces. In this study, almost all of the administered dose was eliminated in the urine after 24 hours (Eckerlin et al. 1987).

As detailed is subsequent subsections, studies regarding histopathology and clinical chemistry data on triclopyr suggest that the liver and kidney 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, 1997). At very high doses, this process may become saturated and triclopyr may interfere/compete with the excretion of other weak acids. Under normal conditions of environmental exposures, however, concentrations of weak acids in the body will be far below those required to saturate the active transport process and this mechanism should not play a substantial or significant role in the assessment of potential health effects. For example, at 5 mg triclopyr/kg bw in dogs, triclopyr is associated with a decrease in phenolsulfonphthalein (PSP) excretion, as standard assay for kidney function. This decrease, however, is associated with competition between triclopyr and PSP rather than a direct toxic effect in the kidney (Finco and Cooper 1995). Conversely, many weak acids also bind to protein and this may inhibit secretion. In the monkey, triclopyr tends to increase the secretion of PSP and other compounds, suggesting that triclopyr may compete with these other compounds for protein binding sites (Timchalk et al. 1997). Again, this competition will be significant only at relatively high doses.

The dermal absorption of triclopyr BEE has been measured *in vitro* using flow-through diffusion cells with skin from rats and humans. After 72 hours, the extent of absorption for un-occluded preparations was 3.7% and 0.7% for rat and human preparations, respectively. Using occluded preparations, the corresponding values increased to 8.6% and 3.3% for rat and human preparations, respectively (Hotchkiss et al. 1992).

These results in experimental mammals and with *in vitro* human skin preparations are consistent with an *in vivo* pharmacokinetics study using volunteers and oral and dermal exposure to triclopyr (Carmichael et al. 1988, 1989). After single oral doses of ^{14}C-labeled triclopyr acid at 0.1 and 0.5 mg/kg, more than 80% of the dose was recovered unmetabolized in the urine within 48 hours. For these oral exposures, the estimated absorption rate coefficients (k_a) were 0.851 $hours^{-1}$ at 0.1 mg/kg and 0.291 $hours^{-1}$ at 0.5 mg/kg.

Dermal exposures consisted of placing 0.65-1.1 mL of Garlon 4 on the forearm so that the applied dose was 5 mg triclopyr/kg body weight. This solution was left on the skin surface for 8 hours and then removed by *"rubbing the dosed area with a paper towel"* (Carmichael et al. 1989, p. 432). Kinetic parameters were determined by measuring triclopyr levels in blood over 12 hours and urine over 84 hours. The average dermal absorption of triclopyr in five volunteers was 1.37% of the applied dose. Based on the pharmacokinetics analysis, the best estimate of the absorption fraction was 1.65%. Presumably, the measured and estimated proportions refer to the total amount of triclopyr recovered in the urine over the 84-hour collection period.

The average absorption halftime for triclopyr, relative to the total amount absorbed, was 16.8 hours [range 11-23 hours in five volunteers]. A dermal absorption coefficient (k_a) of 16.3 $hours^{-1}$ is given in Table 2 of Carmichael et al. (1989). This appears to be an error. The dermal absorption coefficient corresponding to an average halftime of 16.8 hours is 0.041 $hours^{-1}$:

$$t_{\frac{1}{2}} = \frac{\ln(2)}{k}$$

which is very close to the k_a of 0.0448 $hours^{-1}$ given in another publication by the same senior author (Carmichael 1989). This is also very close to the average dermal absorption rate of 0.046 $hour^{-1}$ (range of 0.0163-0.0873 $hour^{-1}$ in 14 individuals) reported by Middendorf (1992). Based on the individual data reported in Carmichael (1989, Table 2, p. 435), the average first-order dermal absorption rate of 0.041 $hour^{-1}$ has a 95% confidence interval of 0.00027 to 0.068 $hour^{-1}$. These values will be used in the current risk assessment for spill scenarios in workers as well as comparable exposure scenarios for the general public (Supplement 2, Worksheet B05).

No data, however, are available on the first-order dermal absorption rate of triclopyr acid. As detailed in the worksheets for triclopyr acid (Supplement 1, Worksheet B03), the first-order dermal absorption rate of triclopyr acid may be estimated from the molecular weight and $K_{o/w}$ as 0.00089 $hour^{-1}$ with a 95% confidence interval of 0.00030 to 0.0026 $hour^{-1}$. While these values could and would be used in the risk assessment in the absence of experimental data, it should be

noted that the corresponding estimates for triclopyr BEE (Supplement 2, Worksheet B03) are 0.0026 (0.0010 to 0.0067) $hour^{-1}$. Based on the central value, these underestimate the experimental first-order dermal absorption rate by a factor of about 16 [0.041 $hour^{-1}$ ÷ 0.0026 = 15.77]. Consequently, there is a reasonable concern that the estimates for triclopyr given in Worksheet B03 of Supplement 1 also underestimate the dermal absorption rate.

In the absence of experimental data on the dermal absorption rate triclopyr acid, the first-order dermal absorption rate of triclopyr acid that is estimated in Worksheet B03 of Supplement 1 – i.e., 0.00089 $hour^{-1}$ with a 95% confidence limit of 0.00030 to 0.0026 $hour^{-1}$ – are multiplied by a factor 16 under the assumption that the algorithm may similarly underestimate the first-order dermal absorption rate. Thus, in Worksheet B05 of Supplement 2, the the first-order dermal absorption rate is set at 0.014 $hour^{-1}$ with a 95% confidence limit of 0.0049 to 0.042 $hour^{-1}$.

3.1.4. Acute Toxicity. Information regarding the acute toxicity of triclopyr and its formulations is summarized in Appendix 4. Like any chemical, triclopyr at sufficiently high exposure levels can cause toxic effects, including death. Nonetheless, triclopyr has a low order of acute lethal potency. In other words, it can be lethal but only at very high doses. Oral doses required to kill 50% of exposed animals (LD_{50} values) range from about 600 to about 1000 mg/kg. There do not appear to be any remarkable differences between the acute oral toxicity of triclopyr free acid (i.e., LC_{50} values of 630 and 729 mg/kg), triclopyr TEA as Garlon 3A (828 mg a.e./kg and 594 mg a.e./kg), and triclopyr BEE (LD_{50} of about 803 mg/kg). The repeated acute dosing study in horses (Osweiler 1983) with involved cumulative doses of up to 1200 mg/kg given over a four day period, resulted in the death of 2/6 ponies and one other animal was euthanized (presumably *in extremis*). This is consistent with the LD_{50} values reported in rats. The primary signs of toxicity in rats include decreased activity and diarrhea. Pathological changes in horses were reported primarily in the liver and kidney.

3.1.5. General Subchronic or Chronic Systemic Toxicity. Systemic toxicity encompasses virtually any effects that a chemical has after the chemical has been absorbed. Certain types of effects, however, are of particular concern to this risk assessment. Such special effects are considered in following subsections and include effects on the nervous system (Section 3.1.6) and immune system (Section 3.1.7), development or reproduction (Section 3.1.8), and carcinogenicity or mutagenicity (Section 3.1.9). This section discusses the remaining studies on systemic toxic effects.

Studies regarding the chronic and subchronic toxicity of triclopyr are summarized in Appendix 5. These include relatively short-term repeated dosing studies conducted as range-finding studies for cancer bioassays (e.g., Tsuda et al. 1987), standard 90-day subchronic studies in rats (Barna-Lloyd et al. 1992), longer-term studies in dogs (e.g., Quast et al. 1977), and lifetime studies in rats (Eisenbrandt et al. 1987) and mice (Tsuda et al. 1987). None of the studies summarized in Appendix 5 are published. These studies were submitted to U.S. EPA/OPP to the support the registration of triclopyr. Full copies of all studies were obtained and reviewed as part of the current risk assessment.

The kidney appears to be the most sensitive target organ for triclopyr, and the dog appears to be the most sensitive species. The lowest effect level for triclopyr is 2.5 mg/kg/day in the dog (Quast et al. 1976, 1977, 1988). In the 1977 study, this dose was associated with decreased phenolsulfonphthalein (PSP) urinary excretion as well as reduced absolute and relative kidney weights. As discussed 3.1.2, the inhibition of PSP excretion can be attributed to competition between triclopyr and PSP for elimination via active anion transport in the proximal tubule cells of the kidney. In the absence of other toxic effects, the 2.5 mg/kg/day dose in the 1977 dog study was classified as a NOEL by U.S. EPA. This determination formed the basis of U.S. EPA's provisional acceptable daily intake of 0.025 mg/kg/day (U.S. EPA 1985) (section 3.3). The NOEL for PBP inhibition in dogs is 0.5 mg/kg/day (Quast et al. 1976).

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

One of the considerations in designating the 2.5 mg/kg/day dose as a NOEL in the earlier study (Quast et al. 1977) was that BUN levels were unaffected. In the later study (Quast et al. 1988), a statistically significant increase in BUN levels were noted in male dogs at 2.5 mg/kg/day (57% increase over pre-exposure levels) and 5.0 mg/kg/day (108% increase over pre-exposure levels). This caused the U.S. EPA to classify the dose of 2.5 mg/kg/day from Quast et al. (1988) as an adverse effect level. At the lowest dose, 0.5 mg/kg/day, BUN levels were elevated by 38% over pre-exposure levels, but this increase was not statistically significant. As discussed in section 3.3., this resulted in the lowering of a provisional U.S. EPA Office of Pesticides RfD to 0.005 mg/kg/day using the 0.5 mg/kg/day dose group as the NOEL for effects on kidney function.

In rodents, kidney effects-hematological and histopathological changes and increased kidney weight—have been observed after subchronic exposure to triclopyr doses as low as 7 mg/kg/day for 90 days (Barna-Lloyd et al. 1992). Damage was characterized as degeneration of the proximal tubules of the kidneys ($\geq$20 mg/kg/day $\cdot$ 90 days) (Landry et al. 1984) and increases in kidney weight (Eisenbrandt et al. 1987, Landry et al. 1984). The highest NOEL below the 7 mg/kg/day AEL for kidney effects in rodents is 5 mg/kg/day for 90 days (Landry et al. 1984).

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

3.1.6. Effects on Nervous System. As detailed in Sections 3.1.6 and 3.1.9, the toxicology of triclopyr has been examined in subchronic, chronic, and multigeneration studies in rodents and in subchronic studies in dogs. In most standard subchronic and chronic rodent bioassays used and accepted by U.S. EPA for pesticide registration brain morphology is assessed. The spinal cord and peripheral nerves (e.g., sciatic nerve) are usually evaluated only if there are other indications of neurotoxicity. The available studies do not report neurological effects at doses that included the maximum tolerated dose.

The toxicity of triclopyr has been studied in various mammalian species (Appendices 4, 5, and 6). A consistent finding at lethal or near-lethal dose levels is lethargy, impaired coordination, weakness, labored respiration, salivation, and tremors, suggesting a neurological component to the toxicity of triclopyr. Similar signs and symptoms are associated with acute exposures to triclopyr acid, triclopyr BEE, and the Garlon formulations. Liver and kidney injury also occurs at dose levels that produce neurological effects; thus, the observed neurological effects may be secondary to toxicity in these organs and related electrolyte or acid/base abnormalities, and/or functional collapse of the cardiovascular system. No other evidence points to a direct effect of triclopyr on the central or peripheral nervous system.

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

3.1.7. Effects on Immune Function. There is very little direct information on which to assess the immunotoxic potential of triclopyr. The only studies specifically related to the effects o triclopyr on immune function are skin sensitization studies conducted on triclopyr BEE and the triethylamine salt of triclopyr (Section 3.1.11). For both of these forms of triclopyr, skin sensitization was observed following standard protocols accepted by the U.S. EPA/OPP (1998a, p. 6). While these studies provide support for asserting that triclopyr may cause skin sensitization, they provide no information useful for directly assessing immune suppressive potential of triclopyr.

As noted in the previous discussion on the neurologic effects (Section 3.1.6), the toxicology of triclopyr has been examined in subchronic, chronic, and multigeneration studies in rodents and in subchronic studies in dogs. None of these studies have reported changes in lymphoid tissues (Appendices 5 and 6).

As detailed in Section 4.1.2.6, the field study by Lochmiller et al. (1995) suggests that the thymus may be affected in rabbits. The thymus has an important role in normal immune function and has a considerable capacity to regenerate (Schuurman et al. 1991). An increase in the size of the thymus could be indicative of repair after injury. Effects on the thymus, however, have not been noted in chronic studies of triclopyr in experimental mammals (Appendix 4). In addition, the lack of a statistically significant difference between rabbits from triclopyr treated areas and rabbits from areas treated only with prescribed burns suggests that the apparent effect may be an

anomaly. For these reasons, the observations made by Lochmiller et al. (1995), albeit noteworthy, are not reason enough to justify a quantitative assessment or to qualitatively identify the thymus as an organ that is particularly sensitive to triclopyr.

3.1.8. Effects on Endocrine Function. In terms of functional effects that have important public health implications, effects on endocrine function would be expressed as diminished or abnormal reproductive performance. This issue is addressed specifically in the following section (Section 3.1.9).

Mechanistic assays are generally used to assess the potential for direct action on the endocrine system. Triclopyr, however, has not been tested for activity as an agonist or antagonist of the major hormone systems (e.g., estrogen, androgen, thyroid hormone). Thus, all inferences concerning the potential effect of triclopyr on endocrine function must be based on inferences from standard toxicity studies. As indicated in the following section (Section 3.1.9), extensive data are available on the reproductive and developmental effects of triclopyr and the current RfD for triclopyr (Section 3.3) is based on a 2-generation reproduction toxicity study in rats (Vedula et al. 1995) with a NOEL of 5.0 mg/kg/day. Although fetal toxicity and abnormalities have been observed at higher doses (Section 3.1.9), there is no indication in this or any other studies (Appendices 5 and 6) that triclopyr caused any of the toxic effects through a mechanism involving endocrine disruption.

3.1.9. Reproductive and Developmental Effects. Toxicity studies for assessing potential adverse effects on reproduction and development fall into two basic categories: multi-generation reproduction studies and teratogenicity studies.

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

Triclopyr has been tested in two multi-generation reproduction studies and several teratogenicity studies (Appendix 6). While abstracts of some studies have been published in the open literature (e.g., Breslin et al. 1996, Breslin and Billington 1995) most of the studies summarized in Appendix 6 are unpublished and the summaries in Appendix 6 are based on the review of full copies of studies received from U.S. EPA/OPP. In some cases, summaries of the studies were

taken from the U.S. EPA/OPP (1998a) RED on triclopyr. These studies are designated by the MRID number appearing before the author citation.

An overview of the studies on the multi-generation reproduction and teratogenicity studies on triclopyr is presented in Table 3-1. This tables includes all of the studies detailed in Appendix 6 and covers triclopyr acid, triclopyr TEA, and triclopyr BEE. At sufficiently high doses, triclopyr can cause adverse reproductive effects as well as birth defects. A consistent pattern with triclopyr, however, as summarized in Table 3-1 and detailed in Appendix 6, is that adverse reproductive effects as well as teratogenic effects occur only at doses that are maternally toxic. At doses which do not cause maternal toxicity, there is not apparent concern for either reproductive or teratogenic effects.

In terms of the risk assessment, the most significant study is the two-generation reproduction study by Vedula et al. (1995). As detailed in Section 3.3, this study is the basis of the current RfD on triclopyr. In this study, male and female rats were exposed to triclopyr in the diet at concentrations resulting in doses of 0, 5, 25, or 250 mg/kg/day, except that the P_1 males in the high dose group were exposed only to concentrations resulting in a daily dose of 100 mg/kg bw/day. The 5 mg/kg/day dose groups evidenced no adverse effects in parents or offspring. At 25 mg/kg/day, degeneration of renal proximal tubules were noted only in adult animals. At 250 mg/kg/day, parental effects included decreased food consumption and body weights as well as histopathologic changes in the liver and kidney. Fetotoxic effects – decreased pup survival and litter sizes – were noted only at 250 mg/kg/day. This dose also resulted in decrease parental fertility. Because no effects were observed at this dose on spermatogenesis or the testes, the decreased fertility was attributed to effects on the female rats. The NOAEL of 25 mg/kg/day for reproductive effects is supported by the study by Hanley et al. (1983), which is also presented as a publication in the open literature (Hanley et al. 1984). This publication reports a three generation reproduction study in the same strain of rats in which no adverse effects were observed on offspring at doses of 3, 10, or 30 mg/kg/day.

At substantially higher doses – i.e., greater than or equal 100 mg/kg/day, triclopyr has been shown to result in birth defects (e.g., Breslin et al. 1996; Bryson 1994b; Hanley et al. 1983; Jones 1995; Thompson et al. 1979). Most of abnormalities have been indicative of delayed growth and have been associated with maternal toxicity. The most severe defects were microphthalmia and anophthamia (small or missing eyes) and other craniofacial abnormalities observed in the Phase I study by Jones (1995) at a dose of 300 mg/kg/day. These effects, however, were not observed in a repeat of this study (Phase II) and similar effects have not been reported in the other teratology studies summarized in Appendix 6. As noted by Jones (1995), the severe abnormalities were in fetuses from dams with the most severe signs of toxicity. Based on the studies by Bryson (1994a) and Breslin and Billinton (1995) with triclopyr BEE and the very similar study by Bryson (1994c) on triclopyr TEA, these two forms of triclopyr appear to be equally toxic, consistent with the basic position adopted by U.S. EPA/OPP (1998a).

3.1.10. Carcinogenicity and Mutagenicity. Information regarding the mutagenicity and carcinogenicity of triclopyr has been reviewed in detail by U.S. EPA (U.S. EPA/OPP 1998a,b,c) as well as in the open literature (Cox 2000). A review of the cancer bioassay data on triclopyr as also been submitted to U.S. EPA in support of the registration of this compound (Goodman and Hildebrandt 1996).

Standard bioassays for carcinogenicity have been conducted in both rats (Eisenbrandt et al. 1987) and mice (Tsuda et al. 1987). Both of these studies are detailed in Appendix 5. 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 – 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 (1998a) has reviewed these studies and determined that the evidence for carcinogenicity is marginal. This position is articulated briefly in the U.S. EPA/OPP (1998a) and, because of the importance of this decision to the risk assessment, the position is worth quoting directly:

> *As a result of the August 9, 1995 meeting of the Agency's Carcinogenicity Peer Review Committee (CPRC), triclopyr was classified as a Group D chemical (not classifiable as to human carcinogenicity). This decision was based on increases in mammary tumors in both the female rat and mouse, and adrenal pheochromocytomas in the male rat, which the majority of the CPRC believed to be only marginal. Overall the majority of the CPRC felt that the animal evidence was marginal (not entirely negative, but yet not convincing). Therefore, the consensus of the CPRC was to classify triclopyr as a Group D chemical, based on what was considered only marginal response and the absence of additional support from structural analogs or genotoxicity.* – U.S. EPA/OPP (1998a, p. 18).

A detailed summary of the mutagenicity studies on triclopyr, most of which are negative, is detailed in Appendix 7.

The discussion of the potential carcinogenicity of triclopyr by Goodman and Hildebrandt (1996) is much more detailed and focuses on a re-evaluation of slides from the original studies as well as an assessment of tumor rates in historical controls. Both types of analyses are common and

appropriate in the assessment of carcinogenicity data. Based on these analyses, Goodman and Hildebrandt (1996) assert that the triclopyr should not be classified as a carcinogen. In terms of the current risk assessment, this position has no impact: The decision by EPA/OPP (1998a) to classify triclopyr as Group D is accompanied automatically by a decision not to derive a cancer potency factor for triclopyr and hence, in terms of a risk assessment, the potential carcinogenicity of triclopyr is not considered quantitatively.

Cox (2000) has suggested that since triclopyr has been shown to cause at statistically significant dose-related increase in mammary gland tumors in both mice and rats, the U.S. EPA guidelines for cancer risk assessment indicate that triclopyr should be classified as a carcinogen. Cox (2000) cites the 1984 guidelines issues by U.S. EPA – i.e., FR 49: 46299-46300. The Agency has since issued additional draft guidelines in both 1996 and 1999, which are available at http://cfpub.epa.gov/ncea/raf/cancer.cfm. The 1984 guidelines do clearly indicate that a compound will be classified as a carcinogen is it has been shown to cause cancer in two species of laboratory animals. The more recent guidelines, however, are less proscriptive and allow the Agency to exercise substantial judgement based on the nature and quality of the data.

Notwithstanding the evolution of the U.S. EPA guidelines for cancer risk assessment, the basic point raised by Cox (2000) is well taken and is the substantial concern to the current risk assessment. Triclopyr has been shown to *cause* the same type of tumors in two species. In addition, while all cancers are a public health concern, the particular tumor type noted in rats and mice (breast cancer) is a common and important form of cancer in humans. Nonetheless, it is worth noting that none of the dose groups in either rats or mice evidenced a statistically significant pair-wise increase in breast tumors. In other words, the magnitude of the response was not substantial. The other important factor discussed by U.S. EPA/OPP (1998a) is the apparent lack of mutagenic activity of triclopyr. As detailed in U.S. EPA/OPP (1998a), only one study – a dominant lethal assay detailed in Appendix 7 – indicated any form of mutagenic activity and the other standard assays for genotoxicity were negative. This is an important point because even if the U.S. EPA had decided to classify triclopyr as a carcinogen, it is plausible that a threshold dose-response assessment would be conducted. In the current risk assessment, a threshold based approach is used for standard toxicity and this approach is based on the most sensitive endpoint – effects on the kidney (Section 3.4).

The only other potentially relevant information encountered in the literature is the epidemiologic report by Gambini et al. (1997). This study analyzes a cohort of rice farmers in Northern Italy, examining mortality patterns. The study attempts to determine whether excess risks for cancer could be detected and associated with chemical exposure. The study finds a significantly lower than expected number of deaths, with a slightly decreased overall cancer mortality. The study indicates that in 1990, 210 kg of triclopyr was used in the region of Italy in which the cohort of rice farmers lived.

This does not entirely alleviate concern for the potential carcinogenic activity of triclopyr. Based on a review of Eisenbrandt et al. (1987) and Tsuda et al. (1987) as well as the discussions in

EPA/OPP (1998a) and Goodman and Hildebrandt (1996), there is no basis for asserting that these studies were seriously flawed.

While that studies by Eisenbrandt et al. (1987) and Tsuda et al. (1987) could be used to derive cancer potency factors, the current risk assessment will defer to the judgment of U.S. EPA/OPP (1998a) and will not quantitatively consider the potential carcinogenic risk of triclopyr. This position is appropriate because the U.S. EPA/OPP (1998a) has conducted a detailed review and it is the U.S. EPA/OPP that has the legislative mandate to determine and regulate the carcinogenic risks from pesticides.

3.1.11. Irritation and Sensitization. As summarized in Appendix 4, exposure to triclopyr formulations may cause irritation to the skin and eyes. Technical grade triclopyr is classified as only slightly irritating (Category IV) (Kuhn 200c). Triclopyr TEA (Garlon 3A) is not a primary skin irritant (Mizell (1988b) but has been shown to cause delayed contact sensitization in some studies (Berdasco 1994a; Mizell 1989) but not in others (Berdasco 1990a,b). Triclopyr BEE has also be shown to cause delayed contact hypersensitivity (Berdasco 1994b). Triclopyr BEE causes more severe skin irritation (Van Beeck and Leegwater 1981a) than triclopyr acid or TEA. This may be due to the more rapid absorption of triclopyr BEE.

Ocular exposure appears to follow a different pattern with triclopyr TEA being much more irritating (Mizell 1988a) than triclopyr acid (Kuhn 2000b) or triclopyr BEE (MRID 40557007 as summarized in U.S. EPA/OPP, 1998a).

3.1.12. Systemic Effects from Dermal Exposure. As discussed in Section 3.1.3, triclopyr appears to be virtually completely absorbed after oral dosing but relatively poorly absorbed after dermal application. Thus, it is to be expected that dermal LD_{50} values are greater than oral LD_{50} values and this appears to be the case. As summarized in Section 3.1.4, oral LD_{50} values in rats range from about 600 to about 1000 mg/kg. As summarized in Appendix 4, dermal LD_{50} values are reported as being over 2000 mg/kg to over 5050 mg/kg. In other words, at these doses, triclopyr killed less than half of the animals. For the studies that were directly reviewed in Appendix 4 – i.e., Kuhn 2000a, Mizell and Lomax 1989, Gilbert 1996, Brooks and DeWildt 2000 – the test animals exhibited no mortality and no clear signs of toxicity other than weight loss.

Repeated dosing studies on triclopyr are summarized in Appendix 8. Three of these studies (Van Beeck and Leegwater 1981a,b; Van Beeck et al. 1984) involve applications of Garlon 4 – i.e., triclopyr BEE. The only study reporting systemic toxic effects is that of Van Beeck et al. 1984 in which rats received dermal doses of 24, 240, and 480 mg a.i./kg bw/day, 5 days per week for 3 weeks. A significant decrease in food intake and growth was observed in males at all dose levels and a significant decrease in food efficiency was observed in males at all dose levels and in females at the highest dose. Based on a review of these and other studies, the U.S. EPA/OPP (1998a) classified the dermal NOAEL for multiple exposures to triclopyr as greater than 1000 mg/kg bw.

3.1.13. Inhalation Exposures. There is very little information regarding the inhalation toxicity of triclopyr. As summarized in Appendix 4, three studies on the inhalation toxicity of triclopyr have been reviewed involving technical grade triclopyr as well as triclopyr BEE and triclopyr TEA. No mortality was observed in any animals. The only study not summarized in U.S. EPA/OPP (1998a) is the recent report by Carter (2000) on technical grade triclopyr. The results of this study – i.e., an LC_{50} of greater than 2.56 mg/L – is essentially equivalent to the reported LD_{50} value of 2.6 mg/L for triclopyr TEA. Based on these results, the U.S. EPA/OPP (1998a) classified inhalation exposures to not be of toxicologic concern.

3.1.14. Adjuvants. Garlon 3A contains the triethylamine salt of triclopyr (44.4%) as well as emulsifiers, surfactants, and ethanol. Garlon 4 contains the butoxyethyl ester (BEE) of triclopyr (61.6%) as well as inerts (38.4%) that include deodorized kerosene.

As reviewed by U.S. EPA/OPP (1998a) triclopyr TEA dissociates extremely rapidly to triclopyr acid and triethanolamine and triclopyr BEE hydrolyzes to triclopyr acid and 2- butoxyethanol. Relatively little information is available on the toxicity of triethanolamine. This compound is classified as a list 3 inert by U.S. EPA/OPP (2001). The List 3 classification reflects the limited toxicity data on triethanolamine and indicates that U.S. EPA/OPP (2001a) was not able to classify this compound as toxic (List 1), potentially toxic (List 2), or essentially non-toxic (Lists 4a or 4b). There is an extensive data base on the toxicity of 2- butoxyethanol and much of the available information relating to potential human health effects has been reviewed by ATSDR (2002). The acute oral MRL for 2- butoxyethanol is 0.4 mg/kg/day and the intermediate MRL for 2- butoxyethanol is 0.07 mg/kg/day (ATSDR 2002). As detailed further in Section 3.3, the acute MRL for 2- butoxyethanol is on the same order as the acute RfD for triclopyr (1 mg/kg/day) and the intermediate MRL for 2- butoxyethanol is similar to the intermediate and chronic RfD for triclopyr (0.05 mg/kg/day). In terms of a practical impact on the risk assessment, the most relevant factor is that both triethanolamine and 2- butoxyethanol will mineralize very rapidly in the environment – i.e., be completely degraded to CO_2. As discussed further in Section 3.2 (Exposure Assessment), this is not the case for triclopyr or TCP, a metabolite of triclopyr. Thus, the uncertainties associated with the toxicity of triethanolamine and the comparable toxicity of 2- butoxyethanol to triclopyr have relatively little impact on this risk assessment. Because triclopyr and the TCP metabolite of triclopyr persist in the environment much longer than triethanolamine or 2- butoxyethanol, it is triclopyr and the TCP metabolite that are the major quantitative focus of the risk assessment. This approach is identical to the position taken in U.S. EPA/OPP (1998a, 2002).

The toxicity of ethanol is extremely well characterized in humans, and the hazards of exposure include intoxication from acute exposure as well as liver cirrhosis and fetal alcohol syndrome (WHO 1988). For chronic exposure, the alcohol contained in Garlon 3A will not be of toxicological significance because of the rapid breakdown of alcohol in the environment and the relatively high levels of alcohol associated with chronic alcohol poisoning. Similarly, alcohol is not likely to pose an acute toxic hazard. Approximately 15 mL of alcohol is contained in 1 oz of an alcoholic beverage containing 50% alcohol (100 proof) [$0.5 \cdot 1$ oz $\cdot$ 29.6 mL/oz $\simeq$ 14.8 mL].

This level may cause mild intoxication in sensitive individuals. Each mL of Garlon 3A contains 0.01 mL of ethanol. Therefore, 1,480 mL, or approximately 1.5 L, of Garlon 3A must be consumed to equal the amount of alcohol contained in 1 oz of an alcoholic beverage. The same amount of Garlon 3A contains 540,000 mg a.e. of triclopyr [1.5 L · 360,000 mg a.e./L]. For a 70 kg man, this dose would equal approximately 770 mg a.e./kg, which is similar to the LD50 for rats. As discussed in the dose-response section (section 3.3), this estimate may be a reasonable approximation of a lethal dose for triclopyr in humans. Thus, compared with the active ingredient, which is triclopyr, the amount of ethanol in Garlon 3A is not toxicologically significant in terms of potential toxicity.

The importance of kerosene in assessing the potential toxicity of Garlon 4 is more difficult to assess. Deodorized kerosene is classified by U.S. EPA as a List 3 Inert. This list contains pesticide inerts that the U.S. EPA considers lacking in toxicological data. The toxicity of kerosene is reviewed in ATSDR (2002). At sufficiently high doses, kerosene can cause many gastrointestinal, central nervous system (CNS), and renal effects. Although some of the effects observed are consistent with the effects (e.g., diarrhea, lethargy, tremors, etc.) observed in mammals given large oral doses of Garlon 4, the same effects are observed in animals given triclopyr alone or Garlon 3A.

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

No monitoring data are available regarding kerosene levels during the application of Garlon 4. Middendorf et al. (1992) monitored triclopyr in air at levels ranging from approximately 5 to 15 µg/m3, based on the personal breathing zone air of workers involved in backpack sprays. If kerosene is present at a concentration of ≤20% in Garlon 4, the corresponding concentration of kerosene in the air would range from approximately 1 to 3 $\mu g/m^3$. The NOAEL for neurological effects in experimental mammals after exposure to kerosene, which ranged from 14 days to 1 year, is approximately 100 mg/m^3; the NIOSH TLV for petroleum distillates is 350 mg/m^3 (ATSDR 2002). Thus, plausible levels of exposure to kerosene during applications of Garlon 4 are approximately 30,000-100,000 below the NOEL for kerosene in experimental mammals and a factor of 120,000-350,000 below the TLV for petroleum distillates. Although some components of kerosene are known to be carcinogenic to humans (e.g., benzene) kerosene is not classified as a carcinogen, and quantitative risk assessments have not been conducted on kerosene (ATSDR 2002).

3.1.15. Impurities and Metabolites. 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. As illustrated in Figure 3-1, TCP is also a metabolite the insecticide chlorpyrifos. While a detailed discussion of the toxicity of chlorpyrifos is beyond the scope of the current document, it is worth noting that chlorpyrifos is an organophosphate insecticide that acts by inhibition of cholinesterase (U.S. EPA/OPP 2001b). As illustrated in Figure 3-1, chlorpyrifos contains the P=S (phosphorus to sulfur double bond) that is characteristic of organothiophosphate cholinesterase inhibitors. This structure is not contained in either TCP or triclopyr and there is no indication that either TCP or triclopyr inhibit cholinesterase.

Nonetheless, TCP is of concern to this risk assessment both because it is a metabolite of triclopyr and because the aggregate risks of exposure to TCP from the breakdown of both triclopyr and chlorpyrifos must be considered. In the RED on triclopyr, the U.S. EPA/OPP (1998a) has considered this issue in some detail (pp. 31 ff). As reviewed by U.S. EPA/OPP (1998a) the chronic toxicity value for TCP is 0.03 mg/kg/day, about the same as the 0.05 mg/kg/day for triclopyr (Section 3.3). For acute exposures, the corresponding values are 30 mg/kg/day for triclopyr and 25 mg/kg/day for TCP. The U.S. EPA estimated dietary exposures at the upper 99.5% level for a young woman – i.e., the most sensitive population in terms of potential reproductive effects, the endpoint of greatest concern for triclopyr. The upper range of acute exposure to triclopyr was estimated at 0.012 mg/kg/day and the upper range of exposure to chlorpyrifos was estimated at 0.016 mg/kg/day. Thus, making the assumption that both triclopyr and chlorpyrifos are totally converted to TCP, the total exposure is about 0.028 mg/kg/day, about a factor of 890 below the level of concern [25 mg/kg/day ÷ 0.028 mg/kg/day = 892.9]. For chronic exposures, the U.S. EPA/OPP (1998a) based on the risk assessment on infants – i.e., individuals at the start of a lifetime exposure. The dietary analysis indicated that the total exposure expressed as a fraction of the RfD was 0.04 for TCP from triclopyr and 0.091 for TCP from chlorpyrifos for a total of 0.131 or a factor of about 7.6 below the level of concern [1÷0.131 = 7.6]. Based on this assessment, the U.S. EPA/OPP (1998a) concluded that:

> *...the existing uses of triclopyr and chlorpyrifos are unlikely to result in acute or chronic dietary risks from TCP. Based on limited available data and modeling estimates, with less certainty, the Agency concludes that existing uses of triclopyr and chlorpyrifos are unlikely to result in acute or chronic drinking water risks from TCP. Acute and chronic aggregate risks of concern are also unlikely to result from existing uses of triclopyr and chlorpyrifos.* – U.S. EPA/OPP (1998a, p. 34).

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

While there is no indication that the general exposures to TCP from the use of triclopyr and chlorpyrifos will result in harmful levels of exposure, this risk assessment does specifically include a consideration of such exposures that may result from specific program activities in the use of triclopyr and chlorpyrifos in forestry applications (Section 3.2.3.7).

3.2. EXPOSURE ASSESSMENT

3.2.1. Overview. Two types of exposure assessments are considered: general and accidental/incidental. The term *general* exposure assessment is used to designate those exposures that involve estimates of absorbed dose based on the handling of a specified amount of a chemical during specific types of applications. The accidental/incidental exposure scenarios involve specific types of events that could occur during any type of application. Based on analyses of several different pesticides using a variety of application methods, default exposure rates are estimated for three different types of applications: directed foliar (backpack), boom spray (hydraulic ground spray), and aerial. These exposure estimates are consistent with and supported by four worker exposure studies involving triclopyr applications.

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

3.2.2. Workers. A summary of the exposure assessments for workers is presented in Table 3-2. Two types of exposure assessments are considered: general and accidental/incidental. The term *general* exposure assessment is used to designate those exposures that involve estimates of absorbed dose based on the handling of a specified amount of a chemical during specific types of applications. The accidental/incidental exposure scenarios involve specific types of events that could occur during any type of application. Details regarding all of these exposure assessments are presented in the worksheets that accompany this risk assessment, as indicated in Table 3-2.

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

for each of these application methods is summarized in Table 3-3. As described in SERA (2001), the ranges of estimated occupational exposure rates vary substantially among individuals and groups, (i.e., by factors of up to 100).

The utility of these general estimates can be evaluated using worker exposure studies involving Garlon 4 (Middendorf 1992; Middendorf et al. 1992; Spencer et al. 2000). Middendorf (1992) assayed exposure in groups of workers applying Garlon 4 by backpack spray to various sites. Middendorf (1992) is a full report and Middendorf et al. (1992) is a summary publication. A summary of relevant data from the full report (Middendorf 1992) is summarized in Table 3-4. Total absorption was determined by the analysis of triclopyr in the urine over a 5-day collection period. The absorption rates, in terms of mg/kg · lb a.i. handled, are summarized in Table 3-4 for three sites reported in Middendorf (1992). As with most studies of this kind, exposure rates among workers varied by a factor of approximately 100. As discussed by Middendorf (1992), a major source of variation appears to involve the use of gloves. Neither of two workers with the highest exposure rates, workers H and I at site 2, wore gloves. All of the workers at site 1, the group with the lowest exposure rate, did ware gloves. As summarized in Table 3-4, the average worker exposure rate from the study by Middendorf (1992) is 0.004 (0.0003 - 0.014) mg/kg · lb a.i. handled. This is very similar to general exposure rates used for backpack workers, 0.003 (0.0003 to 0.01) mg/kg · lb a.i. handled, as summarized in Table 3-3.

The study by Spencer et al. (2000) is a Health and Safety Report from the California EPA that estimates dermal and inhalation exposure of workers who apply Garlon 4 to National Forests (conifer release treatments). Data from this study on the amount handled by each worker, worker body weight, and urinary excretion are summarized in Table 3-5. While the confidence intervals for the worker exposure rates are comparable to those from Middendorf (1992) study at Site 2 and Site 3, the central estimates of exposure are substantially higher. This may be due to the way in which urinary excretion was calculated. While Spencer et al. (2000) attempted to obtain complete urine collections over each 24 hour period, the actual urine collections were highly variable (Spencer et al. 2000, Appendix 1, Table 4), ranging from 30 mL to 1400 mL. To adjust for incomplete urine collection, Spencer et al. (2000) adjusted all urine volumes to 1400 mL. In other words, urinary excretion was calculated as the pooled concentration of triclopyr in the urine multiplied by 1400 mL and divided by the volume of urine collected from the worker. While the 1400 mL urine volume is a reasonable estimate (ICRP 1976), this approach to correcting for incomplete urine collection would tend to over-estimate urinary excretion if the sample was collected during a period high excretion , such as during or shortly after work, but could underestimate exposure if the urine was collected during a period low excretion.

For this risk assessment, the standard exposure rate assumptions specified in Table 3-3 will be used. As noted above, the estimates for backpack workers are very similar to the measured values in the study by Middendorf (1992) and the upper range of the exposure rates – i.e., 0.1 mg/kg/day per lb applied – is only somewhat lower than the rates reported by Spencer et al. (2000). As detailed further in Section 3.4, the relatively minor differences between the exposure

rates from Spencer et al. (2000) and the upper range of exposure rates from Table 3-3 makes very little difference to the assessment of risk.

Worker exposure to triclopyr TEA (as Garlon 3A) has been studied by Abdelghani (1995). This publication, however, does not provide sufficient information to estimate worker exposures as in Tables 3-4 and 3-5. Abdelghani (1995) note that the maximum estimated exposure for any worker was 0.00061 mg/kg bw. As summarized in Worksheet E01, this is consistent with and at the lower range of estimated exposures for workers in this risk assessment.

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

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

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

For this risk assessment, two exposure scenarios are developed for each of the two types of dermal exposure, and the estimated absorbed dose for each scenario is expressed in units of mg chemical/kg body weight. As specified in Table 3-3, the details of these exposure estimates are presented in the worksheets appended to this risk assessment.

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

that is in contact with the surface of the skin and the resulting dermal absorption rate are essentially constant.

For both scenarios (the hand immersion and the contaminated glove), the assumption of zero-order absorption kinetics is appropriate. Following the general recommendations of U.S. EPA/ORD (1992), Fick's first law is used to estimate dermal exposure.

Exposure scenarios involving chemical spills on to the skin are characterized by a spill on to the lower legs as well as a spill on to the hands. In these scenarios, it is assumed that a solution of the chemical is spilled on to a given surface area of skin and that a certain amount of the chemical adheres to the skin. The absorbed dose is then calculated as the product of the amount of the chemical on the surface of the skin (i.e., the amount of liquid per unit surface area multiplied by the surface area of the skin over which the spill occurs and the concentration of the chemical in the liquid) the first-order absorption rate, and the duration of exposure. As summarized in Section 3.1.3, the first-order dermal absorption rates for triclopyr BEE are taken from the study by Carmichael (1989): average first-order dermal absorption rate of 0.041 $hour^{-1}$ has a 95% confidence interval of 0.00027 to 0.068 hour-1. These values are included in Worksheet B05 of Supplement 2 rather than the values calculated in Worksheet B03, which are based on molecular weight and the $k_{o/w}$ for triclopyr. For triclopyr acid, the estimated values given in Worksheet B03 of Supplement 2 are adjusted upward by a factor of 16. The rationale for this approach is also detailed in Section 3.1.3.

For both scenarios, it is assumed that the contaminated skin is effectively cleaned after 1 hour. As with the exposure assessments based on Fick's first law, this product (mg of absorbed dose) is divided by body weight (kg) to yield an estimated dose in units of mg chemical/kg body weight. The specific equation used in these exposure assessments is taken from SERA (2001).

3.2.3. General Public.

3.2.3.1. General Considerations – 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, any number of exposure scenarios can be constructed for the general public, depending on various assumptions regarding application rates, dispersion, canopy interception, and human activity. Several highly conservative scenarios are developed for this risk assessment.

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

The exposure scenarios developed for the general public are summarized in Table 3-6. All exposures are expressed as triclopyr acid equivalents. For most scenarios, the levels of exposure are identical for both triclopyr TEA and triclopyr BEE. The only exceptions are for the scenarios involving dermal exposure and this is due to the assumed differences in dermal exposure rates. Details regarding the assumptions and calculations involved in these exposure assessments are provided in worksheets D01-D09 of the worksheets for triclopyr acid (Supplement 1) and triclopyr BEE (Supplement 2). The remainder of this section focuses on a qualitative description of the data supporting each of the assessments.

3.2.3.2. Direct Spray -- Direct sprays involving ground applications are modeled in a manner similar to accidental spills for workers (see Section 3.2.2.2.). In other words, it is assumed that the individual is sprayed with a solution containing the compound and that an amount of the compound remains on the skin and is absorbed by first-order kinetics. As with the similar worker exposure scenarios, the first-order absorption dermal absorption rate for triclopyr BEE is taken from the study by Carmichael (1989). The dermal absorption rate for triclopyr acid is calculated based on molecular weight and $K_{o/w}$ but adjusted upward by a factor of 16 to account for the potential underestimation of the dermal absorption rate (Section 3.1.3).

For direct spray scenarios, it is assumed that during a ground application, a naked child is sprayed directly with triclopyr. The scenario also assumes that the child is completely covered (that is, 100% of the surface area of the body is exposed), which makes this an extremely conservative exposure scenario that is likely to represent the upper limits of plausible exposure. An additional set of scenarios are included involving a young woman who is accidentally sprayed over the feet and legs. For each of these scenarios, some assumptions are made regarding the surface area of the skin and body weight. These assumptions are detailed and referenced in Worksheet A03 of Supplements 1 and 2.

3.2.3.3. Dermal Exposure from Contaminated Vegetation – In this exposure scenario, it is assumed that the herbicide is sprayed at a given application rate and that an individual comes in contact with sprayed vegetation or other contaminated surfaces at some period after the spray operation. For these exposure scenarios, some estimates of dislodgeable residue and the rate of transfer from the contaminated vegetation to the surface of the skin must be available. No such data are directly available for triclopyr, and the estimation methods of Durkin et al. (1995) are used as defined in worksheet D03. Other estimates used in this exposure scenario involve estimates of body weight, skin surface area, and first-order dermal absorption rates. The estimates of body weight and surface area are detailed in Worksheet A03. The first order dermal absorption rates are identical to those used in the the direct spray scenarios (Section 3.2.3.3).

3.2.3.4. Contaminated Water – Water can be contaminated from runoff, as a result of leaching from contaminated soil, from a direct spill, or from unintentional contamination from aerial applications. For this risk assessment, the two types of estimates made for the concentration of triclopyr in ambient water are acute/accidental exposure from an accidental spill and longer-term

exposure to triclopyr in ambient water that could be associated with the application of this compound to a 10 acre block that is adjacent to and drains into a small stream or pond.

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

The accidental spill scenario assumes that a young child consumes contaminated water shortly after an accidental spill into a small pond. The specifics of this scenarios are given in Worksheet D05. Because this scenario is based on the assumption that exposure occurs shortly after the spill, no dissipation or degradation of triclopyr is considered. This is an extremely conservative scenario dominated by arbitrary variability. The actual concentrations in the water would depend heavily on the amount of compound spilled, the size of the water body into which it is spilled, the time at which water consumption occurs relative to the time of the spill, and the amount of contaminated water that is consumed. Based on the spill scenario used in this risk assessment, the concentration of triclopyr in a small pond is estimated to range from about 0.2 mg/L to 182 mg/L with a central estimate of about 3.6 mg/L (Worksheet D05).

The other acute exposure scenario for the consumption of contaminated water involves runoff into a small stream. Stream monitoring data are available for both triclopyr (Norris et al. 1987) and triclopyr BEE (Kreutzweiser et al. 1995, Smith and McCormack 1988, Thompson et al. 1991, 1995). Details of these studies are summarized in Appendix 11. Peak concentrations in stream water (excluding accidental direct spray), normalized for application rate, range from approximately 0.03 to 0.1 mg/L per kg a.e./ha of application rate or 0.033 to 0.11 mg/L per lb a.e./acre of application rate. The low end of this range is based on applications of triclopyr (0.1 mg/L at 3.4 kg/ha in Norris et al. 1987) and triclopyr BEE (0.056 mg/L at 1.9 kg/ha in Smith and McCormack 1988). The high end of the range is associated with the levels of 0.23-0.35 mg/L after and aerial application of 3.67 kg/ha of Garlon 4. These peak rates are reasonably consistent with what might be expected from simple dilution. For example, an application rate of 1 kg/ha is equivalent to 1,000,000 mg/10,000 m^2 or 100 mg/m^2. Using a stream depth of 0.1-1 m, instantaneous mixing would result in initial maximum concentrations of 100-1,000 mg/m^3 or 0.1-1 mg/L [m^3 = 1,000 L].

While monitoring data provide practical and documented instances of water contamination, monitoring studies may not encompass a broad range of conditions which may occur during program applications – e.g., extremely heavy rainfall. Consequently, for this component of the exposure assessment, the monitored levels in ambient water are compared to modeled estimates based on GLEAMS (Groundwater Loading Effects of Agricultural Management Systems). GLEAMS is a root zone model that can be used to examine the fate of chemicals in various types of soils under different meteorological and hydrogeological conditions (Knisel et al. 1992). The general application of the GLEAMS model to estimating concentrations in ambient water are given in Attachment 3.

For the current risk assessment, the application site was assumed to consist of a 10 acre square area that drained directly into a small pond or stream. The pond dimensions (1000 m^3 or about 0.25 acres with an average depth of 1 meter) are the same as those used in the acute spill scenario. The parameters used for triclopyr in the GLEAMS modeling are summarized in Table 3-7, along with chemical specific parameters for TCP and chlorpyrifos which are discussed below (Section 3.2.3.4.3).

The GLEAMS modeling yielded estimates triclopyr runoff and percolation that were used to estimate concentrations in the stream adjacent to a treated plot, as detailed in Section 5.5 of Attachment 3. The results of the GLEAMS modeling for the small stream are summarized in Table 3-8. These estimates are expressed as the water contamination rate (WCR) - i.e., the concentration of the compound in water in units of mg/L normalized for an application rate of 1 lb a.e./acre.

Overall, the monitoring data are in relatively good agreement with the estimates from GLEAMS. The upper range of the estimates based on monitoring data, 0.11 mg/L per lb applied, is very close to peak rates of 0.054 to 0.243 from the GLEAMS stream modeling for rainfall rates in the range of 50 to 100 inches per year (Table 3-9). The lower range of values from the monitoring data, 0.033 mg/L per lb a.e./acre of application rate, is comparable to peak rates of 0.016 to 0.048 from the GLEAMS stream modeling for rainfall rates in the range of 15 to 25 inches per year (Table 3-9). More or less extreme rainfall rates result in higher and lower estimated concentrations.

Given the close correspondence between the monitoring data and modeling estimates of peak concentrations in stream water, the selection of monitoring data or modeling estimates makes very little difference to the exposure assessment. For this risk assessment, the range of WCR will be taken as 0.001 to 0.4 mg/L per lb applied per acre. The lower range is somewhat arbitrarily set: in very arid environments, no contamination is likely. The upper range of 0.4 mg/L per lb applied is based on the upper range of the modeled stream concentrations from GLEAMS based on clay soil. The typical WCR is taken as 0.09 mg/L per lb applied per acre. This is the geometric mean of the range and the approximate value of maximum concentrations in stream water modeled for all soils at an annual rainfall rate of 50 inches per year.

3.2.3.4.2. LONGER-TERM EXPOSURE – The scenario for chronic exposure to triclopyr from contaminated water is detailed in worksheet D07. This scenario assumes that an adult (70 kg male) consumes 2 liters of contaminated ambient water each day for a lifetime.

As with the above stream scenario, the estimated concentrations in pond water are based on modeled estimates from GLEAMS which are supported by monitoring data. The specific methods used to calculate the concentration of triclopyr in a small pond based on the GLEAMS output are detailed in Section 5.4 of Attachment 3.

The results of the GLEAMS modeling for the pond is summarized in Table 3-9 and the specific estimates of concentrations of triclopyr in ambient water that are used in this risk assessment are summarized in Worksheet B06. As with the corresponding values for a small stream, these estimates are expressed as the water contamination rates (WCR) in units of mg/L per lb applied per acre.

The typical WCR is taken as 30 µg/L or 0.03 mg/L. This is about the average concentration that could be expected at rainfall rates of 20 inches per year in clay or loam soil. The upper limit is taken as 0.05 mg/L, approximately the longer-term average concentration from sandy soil at rainfall rates of 25 inches per year. The lower limit of the WCR is taken as 0.008 mg/L, about the average concentration from clay soil at an annual rainfall rate of 10 inches per year. For comparison, the U.S. EPA/OPP (1998a) estimated 56-day average concentrations for triclopyr at 0.02 mg/L, very close to the typical WCR used in this risk assessment.

3.2.3.4.3. Potential Exposures to TCP – As discussed in Section 3.1.15, the U.S. EPA has conducted extensive analyses on dietary and drinking water exposures to triclopyr and TCP, including the formation of TCP from chlorpyrifos, and concluded that the exposures are far below levels of toxicologic concern. This assessment, however, is based primarily on the agricultural uses of triclopyr – i.e., estimated dietary residues – and does not specifically address potential exposures from forestry applications. In forestry applications, the primary concern would be the formation of TCP as a soil metabolite. TCP is more persistent than triclopyr in soil (Table 3-7) and TCP is relatively mobile in soil (U.S. EPA/OPP 1998a) and could contaminate bodies of water near the site of application. In order to assess the potential risks of TCP formed from the use of triclopyr, the TCP metabolite was modeled along with triclopyr as described in Section 3.2.3.4.1. The results for TCP are summarized in Table 3-10 for a small stream and Table 3-11 for a small pond.

There is very little monitoring data with which to assess the plausibility of the modeling for TCP. As discussed by U.S. EPA/OPP (1998a, p. 65ff), TCP is seldom detected in surface water after applications of triclopyr that result in triclopyr concentrations of up to about 25µg/L, with a limit of detection (LOD) for TCP of 10 µg/L. Thompson et al. (1991) examined the formation of TCP from triclopyr in a forest stream. Consistent with the results reported by U.S. EPA, these investigators failed to detect TCP (LOD=50 µg/L) in stream water with concentrations of triclopyr up to 140 µg/L. This is at least consistent with the GLEAMS modeling of both triclopyr and TCP. As indicated in Table 3-8, the maximum modeled concentrations of triclopyr in stream water range from about 161 to 428 µg/L (for sandy and clay soils respectively) and the corresponding maximum modeled concentration of TCP in stream water (Table 3-10) range from about 5 to 11 µg/L. Thus, given the LOD of 50 µg/L in the study by Thompson et al. (1991), the failure to find TCP in stream water is consistent with the GLEAM modeling.

As discussed by U.S. EPA/OPP (1998a, 2002), TCP is also a metabolite of chlorpyrifos and the U.S. EPA/OPP (1998a, 2002) considered exposures to TCP from both triclopyr and chlorpyrifos in their general dietary and drinking water exposure assessments. While triclopyr and

chlorpyrifos would not be commonly applied together in forestry applications, at least one formulation of chlorpyrifos, Nufos 4E, is labeled for forestry applications and may be applied at a rate of 1 lb/acre for the control of insect pests in tree nurseries and plantations (C&P Press 2002). In order to assess potential exposures to TCP from the application of both triclopyr and chlorpyrifos at the same site, GLEAMS was used to model the application of chlorpyrifos at 1 lb per acre under the same conditions used for triclopyr. The estimated concentrations of TCP in water from such a co-application are summarized in Table 3-12 for a small stream and Table 3-13 for a small pond.

It should be noted that the maximum concentrations for TCP in water reported in Table 3-12 and Table 3-13 do not necessarily reflect simultaneous application of triclopyr and chlorpyrifos. Because triclopyr and chlorpyrifos degrade at different rates, maximum concentration in soil, and hence maximum runoff to water, will occur at different times. Thus, in order to provide the most conservative estimate of exposure to TCP, the maximum concentrations reported in both Table 3-12 and Table 3-13 reflect applications of triclopyr and chlorpyrifos spaced in such a way as to result in the maximum possible concentrations of TCP in water. This extremely conservative approach is discussed further in the risk characterization for both human health (Section 3.4) and ecological effects (Section 4.4).

3.2.3.4.4. Aquatic Weed Control – As noted in Section 2, Garlon 3A may be used in the control of submerged weeds and concentrations of triclopyr in water may not exceed 0.4 mg/L near potable water intakes (Dow AgroSciences 2002; SePRO 2003a). This concentration is identical to the upper range of concentrations in streams (Section 3.2.3.4.1). The, the characterization of plausible risks to humans associated with the use of Garlon 3A for aquatic weed control will be encompassed by the upper range of exposures associated with runoff into small streams.

Higher concentrations of triclopyr in water in areas where Garlon 3A would be directly applied will, of course, be much higher than 0.4 mg/L at lease for a short period of time. Based on the product label (Dow AgroSciences 2002; SePRO 2003a), target concentrations of triclopyr in ambient water for the control of submerged weeds appears to be in the range of 0.75 mg a.e./L to 2.5 mg a.e./L.

How the estimates of concentrations of triclopyr in water were made is not specified in the product label. The product label dose specify application rates of 2 qt/acre to 8 qt/acre. These are equivalent to 1.5 lb a.e./acre to 6 lb a.e./acre which are in turn equivalent to 168.15 mg a.e./m^2 to 872.6 mg a.e./m^2. The initial concentrations in water (assuming instantaneous mixing) would depend on the depth of the water. At a depth of 1 meter, concentrations in the water column would be about 0.17 mg/L to 0.67 mg/L. Thus, the estimates of 0.75 mg a.e./L to 2.5 mg a.e./L appear to anticipate a shallower water depth of about 0.2 to 0.3 meter or 0.6 to 1 foot. This is consistent with the application of Garlon 3A near the shoreline where many aquatic weeds will be most dense.

Thus, concentration of triclopyr in water of 0.75 mg a.e./L to 2.5 mg a.e./L, as specified on the Garlon 3A product label (Dow AgroSciences 2002), appear to be reasonable and will be used to estimate potential acute exposures from the direct application of Garlon 3A to water, similar to the accidental spill scenario (Section 3.2.3.4.1). As specified in Section 3.2.3.4.1, the accidental spill scenario leads to much central estimates of triclopyr in water of about 3.6 mg/L (Worksheet D05), very close to the upper limit of 2.5 mg a.e./L from the direct application of Garlon 3A to standing water for the control of submerged vegetation. Consequently, as discussed further in Section 3.4.3, the central estimates of the accidental spill scenario is used to characterize plausible risks associated with the use of Garlon 3A for the control of submerged vegetation.

3.2.3.5. Oral Exposure from Contaminated Fish – Many chemicals may be concentrated or partitioned from water into the tissues of animals or plants in the water. This process is referred to as bioconcentration. Generally, bioconcentration is measured as the ratio of the concentration in the organism to the concentration in the water. For example, if the concentration in the organism is 5 mg/kg and the concentration in the water is 1 mg/L, the bioconcentration factor (BCF) is 5 L/kg [5 mg/kg ÷ 1 mg/L].

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

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

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

In a field study, no detectable levels of triclopyr were found in fish after an initial application rate of 2.5 mg a.e./L as Garlon 3A. Modest levels of bioconcentration, however, were noted in crayfish and clams (BCF $\leq$4 L/kg) with rapid decreases in tissue levels as water levels decreased (Woodburn et al. 1993b).

For this risk assessment, the BCF values are taken from Rick et al. (1996) based on total residues. As detailed further in the risk characterization, this approach is used to consider the potential effects of both triclopyr and TCP.

For both the acute and longer-term exposure scenarios involving the consumption of contaminated fish, the water concentrations of triclopyr used are identical to the concentrations used in the contaminated water scenarios (see Section 3.2.3.4). The acute exposure scenario is based on the assumption that an adult angler consumes fish taken from contaminated water shortly after an accidental spill of 200 gallons of a field solution into a pond that has an average depth of 1 m and a surface area of 1000 m^2 or about one-quarter acre. No dissipation or degradation is considered. Because of the available and well-documented information and substantial differences in the amount of caught fish consumed by the general public and native American subsistence populations (U.S. EPA 1996), separate exposure estimates are made for these two groups, as illustrated in worksheet D08. The chronic exposure scenario is constructed in a similar way, as detailed in worksheet D09, except that estimates of triclopyr concentrations in ambient water are based on GLEAMS modeling as discussed in Section 3.2.3.4.

3.2.3.6. Oral Exposure from Contaminated Vegetation – Under normal circumstances and in most types of applications, it is extremely unlikely that humans will consume vegetation contaminated with triclopyr. Any number of accidental scenarios could be developed involving either spraying of crops, gardens, or edible wild vegetation. Again, in most instances and particularly for longer-term scenarios, treated vegetation would probably show signs of damage from exposure to triclopyr (Section 4.3.2.4), thereby reducing the likelihood of consumption that would lead to significant levels of human exposure.

Notwithstanding that assertion, it is conceivable that individuals could consume contaminated vegetation that is accidentally sprayed. One of the more plausible scenarios involves the consumption of contaminated berries after the accidental spray of an area in which wild berries grow. The most relevant publication for assessing exposure from such a scenario is that of Siltanen et al. (1981). These investigators monitored levels of triclopyr on cowberries and bilberries after backpack sprays of Garlon 3A at application rates of 0.25, 0.75, and 2.25 kg a.e./ha [0.22, 0.67, and 2 lbs/acre]. The data on residue rates – i.e., ppm in fruit per lb/acre of triclopyr applied – are illustrated in Figure 3-2. Although there is substantial scatter in the data (r^2=0.62), there is no consistent deviation from the simple first order dissipation model. The estimated residues at time zero is 1.85 ppm with a 95% confidence interval of 1.23 to 2.77 ppm. The decay coefficient (ke) is equal to 0.0184 $days^{-1}$ with a 95% confidence interval of 0.010 to 0.027 $days^{-1}$, corresponding to halftimes of 37.7 (25.7 to 69.3) days.

Typically, Forest Service risk assessments will use empirical relationships between application rate and residues on vegetation. These are illustrated in Worksheet A04 of Supplements 1 and 2. Consistent with the approach taken by U.S. EPA, Forest Service risk assessments use the more recent set of residue rates from Fletcher et al. (1994), rather than the earlier residue rates derived by Hoerger and Kenaga (1972). As indicated in Worksheet A04, however, residue rates for fruit

from Fletcher et al. (1994) are 7 ppm per lb/acre with an upper range of 15 ppm per lb/acre. The rates recommended by Hoerger and Kenaga (1972) are 1.5 ppm per lb/acre with an upper range of 7 ppm per lb/acre. The study by Siltanen et al. (1981) is consistent with the estimates by Hoerger and Kenaga (1972) but not those of Fletcher et al. (1994). Given the direct relevance of the Siltanen et al. (1981) study, the central and upper ranges of residue rate from this study – i.e., 1.85 ppm and 2.77 ppm will be used rather than either of the two sets of standard rates for this exposure scenario as well as for other exposure scenarios involving the consumption of contaminated fruit.

The two accidental exposure scenarios developed for this exposure assessment include one scenario for acute exposure, as defined in Worksheet D03 and one scenario for longer-term exposure, as defined in Worksheet D04. In both scenarios, the concentration of triclopyr on contaminated vegetation is estimated using the empirical relationships between application rate and concentration on vegetation from the Siltanen et al. (1981) data illustrated in Figure 3-2. For the acute exposure scenario, the estimated residue level is taken as the product of the application rate and the residue rate (Worksheet D03).

For the longer-term exposure scenario (D04), a duration of 90 days is used and the dissipation on the vegetation is estimated using the halftime 37.7 (25.7 to 69.3) days from the data of Siltanen et al. (1981). Although the duration of exposure of 90 days is somewhat arbitrarily chosen, this duration is intended to represent the consumption of contaminated fruit that might be available over one season. Longer durations could be used for certain kinds of vegetation but would lower the estimated dose (i.e., would result in a less conservative exposure assessment).

3.3. DOSE-RESPONSE ASSESSMENT

3.3.1. Overview. Generally, the dose-response assessments used in Forest Service risk assessments adopt RfDs proposed by the U.S. EPA as indices of acceptable exposure. An RfD is basically defined as a level of exposure that will not result in any adverse effects in any individual. The U.S. EPA RfDs 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 U.S. EPA recommends a chronic RfD of 0.05 mg/kg/day. This chronic RfD is based on the two-generation reproduction study in rats in which degeneration of renal proximal tubules were noted in adult animals at a dose of 25 mg/kg/day but not at 5 mg/kg/day. The 5 mg/kg/day NOAEL dose was divided by 100, a factor of 10 to account for uncertainties in species-to-species extrapolation and another factor of 10 to encompass sensitive individuals in the population. Thus, the resulting RfD is 0.05 mg/kg/day. Under the Food Quality Protection Act (FQPA), the U.S. EPA is required to evaluate whether or not an additional uncertainty factor is required for the protection of children. Because the parental NOAEL for reproduction studies are below any adverse reproductive effects, the U.S. EPA has determined that no additional FQPA uncertainty

factor is required. The U.S. EPA has recommended an acute RfD for triclopyr of 1 mg/kg/day for the general population. This is based on the NOAEL of 100 mg/kg/day from at teratogenicity study (i.e., a study test the potential for the development of birth defects). This acute RfD is not applicable to females between the ages of 13-50 years – i.e., females of child bearing age. For these individuals, the acute RfD is set at 0.05 mg/kg/day, equivalent to the chronic RfD.

The U.S. EPA has not derived a formal RfD for TCP, the metabolite of triclopyr. For the current risk assessment, the risk values used for risk characterization are identical to the most recent and conservative risk values proposed by U.S. EPA: 0.025 mg/kg/day for acute exposures and 0.012 mg/kg/day for chronic exposures. The acute value is based on a developmental toxicity study in rabbits with NOAEL of 25 mg/kg/day and an uncertainty factor of 1000. The chronic risk value is based on a 12 mg/kg/day NOAEL, also using an uncertainty factor of 1000.

3.3.2. Triclopyr. Generally, the dose-response assessments used in Forest Service risk assessments adopt RfDs proposed by the U.S. EPA as indices of acceptable exposure. An RfD is basically defined as a level of exposure that will not result in any adverse effects in any individual. The U.S. EPA RfDs 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.

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

Under the Food Quality Protection Act (FQPA), the U.S. EPA is required to evaluate whether or not an additional uncertainty factor is required for the protection of children. As summarized in Table 3-1, the parental NOAEL of 5 mg/kg/day is below any adverse reproductive effects. Consequently, the U.S. EPA/OPP (1998a, 2002) has determined that no additional FQPA uncertainty factor is required.

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. This 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 number of live fetuses, increased number of fetal deaths, and increased number of fetal and/or litter incidence of skeletal anomalies and

variants. As also summarized in Table 3-1, the 30 mg/kg/day NOEL is supported by a number of other teratogenicity studies as well as the multi-generation reproduction study by Beliles and Wosu 1976.

In the most recent pesticide tolerance for triclopyr (U.S. EPA/OPP 2002), the U.S. EPA/OPP has recommended an explicit acute RfD of 1 mg/kg/day for the general population. This appears to be based on the NOAEL of 100 mg/kg/day from the study by Jones (1995) in which rats were administered gavage doses of triclopyr BEE at 0, 30, 100, or 300 mg/kg/day on days 6 through 15 of gestation. At 300 mg/kg/day, toxic responses included signs of marked maternal toxicity including four deaths, overt clinical signs in a few dams, mean body weight loss and decreased mean body weight gain, decreased mean feed consumption, increased mean water consumption, and increased mean liver and kidney weights. In addition, fetal effects included both skeletal and soft-tissue malformations. This acute RfD is not applicable to females between the ages of 13-50 years – i.e., of child bearing age. For these individuals, the U.S. EPA/OPP (2002) recommends an acute RfD of 0.05 mg/kg/day, equivalent to the chronic RfD.

Previously, the U.S. EPA/OPP (1995) had derived an RfD based on the study by Quast et al. (1988) in which the triclopyr triethylamine salt was administered in the diet to dogs at levels that resulted in daily doses of 0.5, 2.5, or 5.0 mg/kg/day over a 1-year period. The two higher doses were classified as adverse effect levels based on dose-related increases in serum urea nitrogen and creatinine, indicative of decreased glomerular filtration. The lowest dose was classified as a NOAEL. As summarized in Appendix 5, there is a dose-dependent increase in BUN in male rats at all dose levels: 38% at 0.05 mg/kg/day, 57% at 2.5 mg/kg/day, and 208% at 5 mg/kg/day. The low dose is regarded as a NOAEL because the increase, relative to pre-exposure levels was not statistically significant. Because of differences between primates and dogs (e.g., Timchalk and Nolan, 1997) in the pharmacokinetics of triclopyr as well as other weak acids, the dog is not considered an appropriate model for human risk assessment and this lower RfD is not recommended by U.S. EPA and is not used in the current risk assessment.

For risk characterization, the current risk assessment will adopt the most recent RfD values recommended by U.S. EPA – i.e., 1 mg/kg for acute exposures in the general population and 0.05 mg/kg/day for exposure scenarios of one month to a lifetime. Also consistent with the approach taken by U.S. EPA/OPP (2002), the acute RfD of 1 mg/kg/day will be applied to the general population but not to women of child-bearing age.

3.3.3. TCP. As discussed in Section 3.1.15, TCP is of concern to the human health risk assessment both because it is a metabolite of triclopyr and because the aggregate risks of exposure to TCP from the breakdown of both triclopyr and chlorpyrifos must be considered.

While the U.S. EPA has not derived a formal RfD for TCP, the RED on triclopyr (U.S. EPA/OPP 1998a, pp. 31 ff) as well as the RED on chlorpyrifos (U.S. EPA/OPP 2001b) use a chronic value of 0.03 mg/kg/day for the risk characterization for TCP. In the more recent pesticide tolerances for triclopyr (U.S. EPA/OPP 2002, pp. 58722), a somewhat lower value is

used for the risk characterization of TCP: a dose of 0.012 mg TCP/kg/day derived using an uncertainty factor of 1000 and data from a chronic study in dogs in which changes in clinical chemistry at a dose of 48 mg/kg/day (LOAEL) but no effects at 12 mg/kg/day (NOAEL).

For acute effects, the pesticide tolerances for triclopyr (U.S. EPA/OPP 2002, pp. 58722) use an acute value of 0.025 mg/kg/day based on a developmental toxicity study in rabbits with NOAEL of 25 mg/kg/day and a corresponding LOAEL of 100 mg/kg/day in which an increased incidence of hydrocephaly and dilated ventricles were noted in rabbits.

For both acute and chronic exposures the uncertainty factor for TCP is set at 1000. This value is comprised of the factors of 10 to account for uncertainties in species-to-species extrapolation and another factor of 10 to encompass sensitive individuals in the population as well a an additional factor of 10 for the potentially higher sensitivity of children – i.e., the FQPA uncertainty factor.

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

3.4. RISK CHARACTERIZATION

3.4.1. Overview. There is no indication that workers will be subject to hazardous levels of triclopyr at the typical application rate of 1 lb/acre and under typical exposure conditions. Nonetheless, at the upper range of exposures, all application methods exceed the level of concern based on the chronic RfD but not the acute RfD. Thus, for workers who may apply triclopyr repeatedly over a period of several weeks or longer, it is important to ensure that work practices involve reasonably protective procedures to avoid the upper extremes of potential exposure. At higher application rates, particularly rates that approach the maximum application rate of 10 lbs/acre, measures should be taken to limit exposure. These measures would need to be developed on a case-by-case basis depending on the specific application rates that are used and the type of the applications that are employed.

For members of the general public, the risk characterization is thus relatively unambiguous at the typical application rate of 1 lb/acre: based on the available information and under the foreseeable conditions of exposure, there is no route of exposure or exposure scenario suggesting that the general public will be at risk from longer-term exposure to triclopyr. Even at the maximum projected application rate of 10 lbs/acre, the only longer-term scenario that exceeds the level of concern is the consumption of contaminated fruit This is a standard scenario used in all Forest Service risk assessments and is extremely conservative – i.e., it assumes that fruit that has been directly sprayed is harvested and consumed for a prolonged period of time and that the contaminated fruit accounts for 100% of the individuals consumption of fruit. Under these extreme conditions, the level of concern (a hazard quotient of unity) is exceeded by a factor of 5 at the upper range but not the central estimate of exposure. Several acute exposures also lead to hazard quotients that are above the level of concern at the upper range of exposure. Two dermal exposures to triclopyr BEE – i.e., accidental spray of a woman over the lower legs as well as

dermal contact with contaminated vegetation by a woman – exceed the level of concern at the central estimate of exposure. The use of the highest application under consideration – i.e., 10 lbs/acre – alters the risk characterization for acute exposures terms of dermal exposures and the spill into a pond. At an application rate of 10 lbs/acre, both triclopyr BEE and triclopyr TEA formulations would exceed the level of concern for all dermal exposure scenarios at the upper range of exposure as well as some central estimates of exposure. Again, all of these dermal exposure assessments are extremely conservative and designed to identify which possible types of exposure would be most hazardous. For triclopyr, such scenarios include dermal contact and accidental spills into water.

The U.S. EPA (1998a, 2002) has conducted extensive analyses of dietary exposure to TCP from the use of triclopyr as well as the aggregate risks from exposure to TCP from the use of both triclopyr and chlorpyrifos. While these dietary exposures appear to be substantially below a level of concern, the risk assessment by EPA does not specifically address concerns for contamination of water with TCP as a soil metabolite of triclopyr and chlorpyrifos. As part of the current risk assessment, exposures to TCP based on modeling of water contamination from the application of both triclopyr and chlorpyrifos indicate that the peak exposure to TCP in water is below the concentration associated with the chronic risk value for TCP. Thus, there is no basis for asserting that the use of triclopyr with or without the use of chlorpyrifos will result in hazardous exposures of humans to TCP.

3.4.2. Workers. The risk characterization for workers is summarized in Table 3-14. In this as well as in similar tables for the general public, risk is characterized quantitatively as the hazard quotient, the estimated exposure divided by the appropriate RfD.

In Table 3-14, two sets of hazard quotients are presented for general exposures – i.e., the total exposure that a worker might receive during directed ground, broadcast ground, and aerial applications – at an application rate of 1 lb/acre. One set of hazard quotients is based on the chronic RfD of 0.05 mg/kg/day and the other set of hazard quotients is based on the acute RfD. The use of both acute and chronic RfD for general occupational exposures is atypical for Forest Service risk assessments and is used here for at least conceptual consistency with U.S. EPA/OPP (1998a) and in order to more fully elaborate potential worker risks under different conditions.

In Table 3-14, as well as in Table 3-15 (risk characterization for the general public), the hazard quotients are the level of exposure divided by the RfD then rounded to one or two significant decimal places or digits. Hazard quotients >1 and ≤ 2 are shown to two significant digits. All others are rounded to one significant decimal place or integer. All hazard quotients that are below the level of concern – i.e., a hazard quotient below unity – are expressed in scientific notation. All hazard quotients greater than unity are expressed in fixed point decimal notation and highlighted with a shaded background.

As detailed in the RED, U.S. EPA/OPP (1998a) elected not to conduct risk characterizations for workers. The following rationale is given by U.S. EPA/OPP (1998a):

> *No short- or intermediate-term risk assessment was required for handler exposures to triclopyr because no toxicological endpoints of concern were identified in a 21 day dermal toxicity study in rabbits at the highest dose (1000 mg/kg/day) indicating very low toxicity via the dermal route of exposure. ... At this time, no chronic risk assessment is required for handler exposures to triclopyr, since none of the current handler exposure scenarios is likely to result in chronic exposure.* – U.S. EPA/OPP (1998a, p. 28).

The basis for asserting that *chronic exposure* is unlikely is not clear and may somewhat depend on the definition of the term *chronic*. Clearly, occupational exposures will not occur over a full lifetime. Nonetheless, as discussed in Section 3.3.2, the U.S. EPA/OPP (2002) applies the *chronic* RfD to exposures ranging from exposure periods of one to six months up to lifetime exposures. In addition, for dermal exposures, the U.S. EPA/OPP (2002) appears to characterize risk based on the oral RfD and assumptions concerning dermal exposure. This is similar to the general approach that has been used in Forest Service risk assessments.

Thus, for the current risk assessment, the use of the acute RfD for risk characterizations of general worker exposures is intended only to illustrate the consequences of applying triclopyr sporadically as part of other activities. As with past Forest Service risk assessments, the basic risk characterization will be based on the chronic RfD.

Table 3-14 also includes two sets of accidental/incidental dermal exposures, one for the triclopyr TEA and the other for triclopyr BEE. This approach is necessary because, as discussed in Section 3.1.3, formulations containing triclopyr BEE may be more rapidly absorbed than formulations containing triclopyr TEA.

As indicated in Table 3-14, central estimates of the hazard index based on the chronic RfD are below the level of concern (unity) for all application methods at an application rate of 1 lb/acre. As indicated in Section 2.4, this is the typical application rate that will be used in Forest Service Programs. At the upper ranges of exposure, however, all general exposures result in hazard quotients that are above unity – i.e., values of 1.6 to 3. As detailed in Section 3.3.2, the RfD is based on a NOAEL of 5 mg/kg/day. The associated LOAEL is a factor of 5 above the NOAEL (25 mg/kg/day) and, at the LOAEL, kidney damage was noted – i.e., degeneration of renal proximal tubules. Thus, the projected hazard quotients are in a region above the adjusted NOAEL but below the LOAEL.

The above hazard quotients apply only to the typical application rate of 1 lb/acre. As also indicated in Section 2.4, the highest application rate considered in this risk assessment is 10 lbs/acre. At this application rate, all of the hazard quotients for general exposures would be

multiplied by a factor of 10. Thus, the central estimates of exposure would be associated with generally hazard quotients that are above the level of concern – i.e., hazard quotient of 3 to 4 – and the upper ranges of exposure would be associated with hazard quotients in the range of 16 to 30. Since the ratio of the LOAEL to NOAEL in the study used to derived the RfD is a factor of 5, hazard quotients that approach or exceed a factor of 5 could be associated with adverse effects on the kidney. While these might not lead to frank signs of toxicity that would be detectable in workers, they are nonetheless undesirable.

Accidental exposures of workers to formulations containing triclopyr TEA do not lead to hazard quotients that exceed a level of concern based on the acute RfD of 1 mg/kg/day. As noted in Section 3.3, however, this acute RfD is not applied to women of child bearing age and the chronic RfD of 0.05 mg/kg/day is used. Thus, for female workers, the level of concern would be 0.05 rather than unity. Even with this more conservative criterion, none of the hazard quotients for accidental scenarios for triclopyr TEA formulations exceed a level of concern at the central estimate of exposure. At the upper limits of potential exposure, the hazard quotient for triclopyr TEA exceeds 0.05 for both spill scenarios. Triclopyr BEE formulations present a greater risk. The level of concern for the general population is exceeded at the upper range of exposure for wearing contaminated gloves for 1 hour. For female workers, the level of concern is exceeded at the upper range of exposure for all accidental scenarios and the level of concern is reached at the central estimate of exposure for wearing contaminated gloves for 1 hour.

The verbal interpretation of these hazard quotients is somewhat ambiguous. Under typical conditions of application and at the typical application rate of 1 lb/acre, there is no indication that workers will be subject to hazardous levels of triclopyr. Nonetheless, at the upper range of exposures, all application methods exceed the level of concern based on the chronic RfD but not the acute RfD. Thus, for workers who may apply triclopyr repeatedly over a period of several weeks or longer, it is important to ensure that work practices involve reasonably protective procedures to avoid the upper extremes of potential exposure.

At higher application rates, particularly rates that approach the maximum application rate of 10 lbs/acre, measures should be taken to limit exposure. These measures would need to be developed on a case-by-case basis depending on the specific application rates that are used and the type of the applications that are employed.

3.4.3. General Public. The risk characterization for the general public is summarized in Table 3-15. As with workers, risk is characterized quantitatively as the hazard quotient, the estimated exposure divided by the appropriate RfD. Also as with workers, all hazard quotients for longer-term exposure are based on the chronic RfD of 0.05 mg/kg/day. For acute exposures involving a child or man, the hazard quotients are based on the acute RfD of 1 mg/kg/day for the general population. The acute RfD is not used for women of child bearing age and all hazard quotients for acute exposure involving a woman are based on the chronic RfD of 0.05 mg/kg/day. As discussed in Section 3.3, the U.S. EPA/OPP (2002) recommends this approach for women of

child bearing age. Note that all hazard quotient for a woman in Table 3-15 are derived from the RfD of 0.05 mg/kg/day and thus the level of concern is unity.

None of longer-term exposure scenarios exceed a level of concern. Although there are several uncertainties in the longer-term exposure assessments for the general public, as discussed in Section 3.2.3, the upper limits for hazard indices are below a level of concern by factors of about 2 (longer term consumption of contaminated fruit) to over 100,000 (longer-term consumption of fish by the general population). The risk characterization is thus relatively unambiguous: based on the available information and under the foreseeable conditions of exposure, there is no route of exposure or exposure scenario suggesting that the general public will be at risk from longer-term exposure to triclopyr at the typical application rate of 1 lb/acre.

Even at the maximum projected application rate of 10 lbs/acre, the only longer-term scenario that exceeds the level of concern is the consumption of contaminated fruit. This is a standard scenario used in all Forest Service risk assessments and is extremely conservative – i.e., it assumes that fruit that has been directly sprayed is harvested and consumed for a prolonged period of time and that the contaminated fruit accounts for 100% of the individuals consumption of fruit. Under these extreme conditions, the level of concern (a hazard quotient of unity) is exceeded – i.e., a hazard quotient of 5.

Several acute exposures also lead to hazard quotients that are above the level of concern at the upper range of exposure. Two dermal exposures to triclopyr BEE – i.e., accidental spray of a woman over the lower legs as well as dermal contact with contaminated vegetation by a woman – exceed the level of concern at the central estimate of exposure.

The use of the highest application under consideration – i.e., 10 lbs/acre – alters the risk characterization for acute exposures terms of dermal exposures and the spill into a pond. At an application rate of 10 lbs/acre, both triclopyr BEE and triclopyr TEA formulations would exceed the level of concern for all dermal exposure scenarios at the upper range of exposure as well as some central estimates of exposure. Again, all of these dermal exposure assessments are extremely conservative and designed to identify which possible types of exposure would be most hazardous. For triclopyr, such scenarios include dermal contact and accidental spills into water.

3.4.4. TCP. As discussed in Section 3.1.15, the U.S. EPA (1998a, 2002) has conducted extensive analyses of dietary exposure to TCP from the use of triclopyr as well as the aggregate risks from exposure to TCP from the use of both triclopyr and chlorpyrifos. While dietary exposures appear to be substantially below a level of concern, the risk assessment by EPA does not specifically address concerns for contamination of water with TCP as a soil metabolite of triclopyr and chlorpyrifos. As detailed in Section 3.2.3.4.3, concentrations of TCP in a small stream could reach up to 11 ppb from the use of triclopyr at a rate of 1 lb/acre (Table 3-10) and up to 68 ppb in a small stream from the use of triclopyr at a rate of 1 lb/acre and chlorpyrifos at a rate of 1 lb/acre (Table 3-12). Much lower peak concentrations would be expected in small ponds.

As detailed in Section 3.3.3, the RfD for TCP used by U.S. EPA (2002) is 0.012 mg/kg/day for chronic exposure and 0.025 mg/kg/day for acute exposure. The child is the most exposed individual, consuming 1L of water per day at a body weight of 10 kg (Worksheet A03). Thus, based on the chronic RfD of 0.012 mg/kg/day, the associated concentration in water would be 0.12 mg/L or ppm [0.012 mg/kg/day × 10 kg/1 L/day] which is in turn equivalent to 120 ppb. Since the peak exposure to TCP in water is below the concentration associated with the chronic RfD, there is no basis for asserting that the use of triclopyr with or without the use of chlorpyrifos will result in hazardous exposures of humans to TCP.

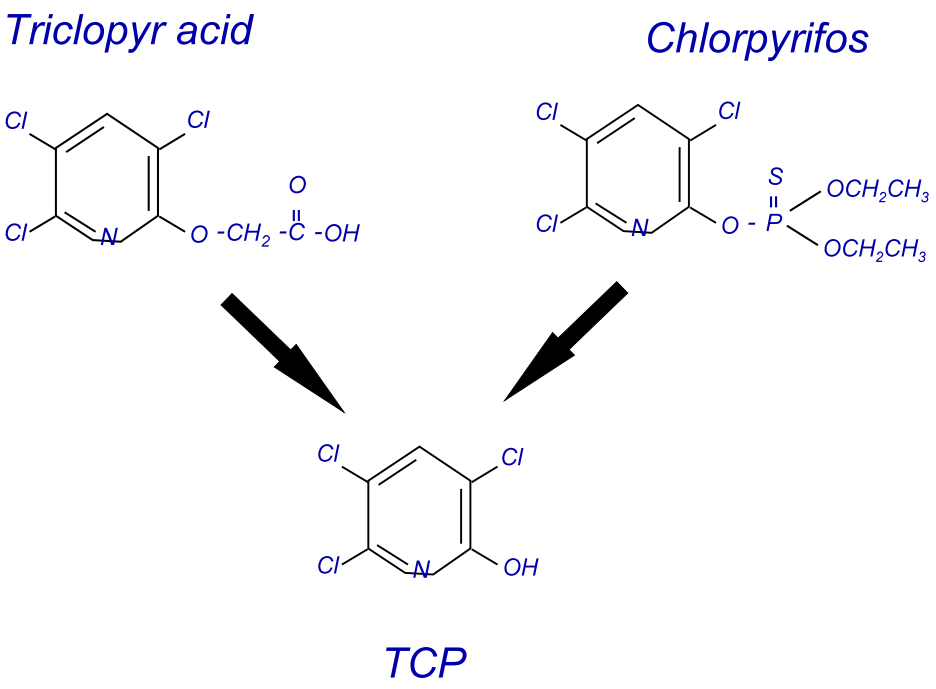


Figure 3-1: Structures TCP, Triclopyr, and Chlorpyrifos.

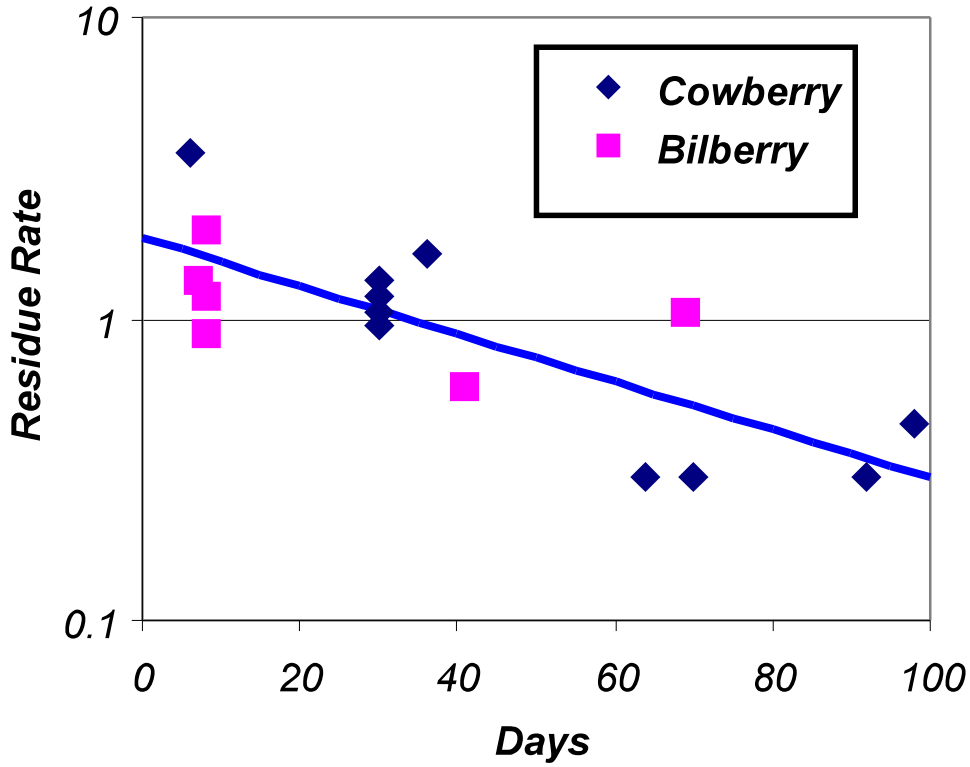


Figure 3-2: Residues of triclopyr (ppm per lb/acre) on cowberries and bilberries (data from Siltanen et al. 1981).

Table 3-1: Summary of studies on the reproductive effects of triclopyr acid, triclopyr TEA, and triclopyr BEE [*].

Type	Parental		Offspring		Reference
	NOAEL	LOAEL	NOAEL	LOAEL	
Multi-generation	5	25	25	250	Vedula et al. 1995
	30		30		Beliles and Wosu 1976
Terato-genicity, Rats		50	100	200	Thompson et al. 1979
		50	100	200	Hanley et al. 1983
	100	300	100	300	Bryson 1994b
		50	100	200	Breslin 1990a
	30	100	100	300	Breslin et al. 1996
	5	30	100	300	Breslin et al. 1996
		30	30	100	Jones 1995, Phase I
	5	30	100	300	Jones 1995, Phase II
Terato-genicity, Rabbits		25	100		Smith et al. 1960
	10	25	25		Hanley et al. 1983
	30	100	30	100	Bryson 1994a [BEE]
	30	100	30	100	Breslin and Billington 1995 [BEE]
	30	100	30	100	Bryson 1994c [TEA]
	25	75	75		Kirk et al. 1989
	10	30	100		Breslin and Billington 1995 [TEA]

[*] See Appendix 6 for details. Triclopyr acid unless otherwise specified. TEA = triethylamine salt. BEE= butoxyethyl ester.

Table 3-2: Summary of Worker Exposure Scenarios for an Application Rate of 1 lb/acre.

Scenario	Dose (mg/kg/day or event)			Exposure Assessment Worksheet
	Central	Lower	Upper	
General Exposures (dose in mg/kg/day)				
Directed ground spray (Backpack)	1.31e-02	4.50e-04	8.00e-02	C01a
Broadcast ground spray (Boom spray)	2.24e-02	6.60e-04	1.51e-01	C01b
Aerial applications	1.47e-02	2.40e-04	8.00e-02	C01c
TEA - Accidental/Incidental Exposures (dose in mg/kg/event)				
Immersion of Hands, 1 minute	2.30e-05	6.60e-06	2.59e-04	C02a
Contaminated Gloves, 1 hour	1.38e-03	3.96e-04	1.56e-02	C02b
Spill on hands, 1 hour	6.41e-03	1.41e-03	9.26e-02	C03a
Spill on lower legs, 1 hour	1.58e-02	3.47e-03	2.28e-01	C03b
BEE - Accidental/Incidental Exposures (dose in mg/kg/event)				
Immersion of Hands, 1 minute	7.87e-03	2.64e-03	7.20e-02	C02a
Contaminated Gloves, 1 hour	4.72e-01	1.58e-01	4.32e+00	C02b
Spill on hands, 1 hour	1.85e-02	7.78e-05	1.51e-01	C03a
Spill on lower legs, 1 hour	4.56e-02	1.92e-04	3.73e-01	C03b

[1] Worksheets for TEA refer to Supplement 1 of the risk assessment and worksheets for BEE refer to Supplement 2 of the risk assessment.

Table 3-3: Occupational exposure rates used in risk assessments [a]			
Worker Group	Rate (mg/kg bw per lb applied)		
	Central	Lower	Upper
Directed foliar	0.003	0.0003	0.01
Broadcast foliar	0.0002	0.00001	0.0009
Aerial	0.00003	0.000001	0.0001
[a] Taken from SERA (2001).			

Table 3-4: Estimated absorption rates for workers involved in backpack applications of triclopyr BEE as Garlon 4 from the study by Middendorf (1992).

Worker	Amount Handled (lb a.e.)[a]	Body Weight (kg)[b]	Amount Absorbed (mg)[b]	Exposure Rate (mg/kg bw per lb a.e.)
		Site 1 (all wore gloves)		
A[c]	4.8	91.2	0.065	0.00015
B	4.8	83.3	0.259	0.00065
C	4.8	93.2	0.697	0.00156
D	4.8	78.3	1.902	0.00506
	Geometric mean (95% C.I.):			0.0009 (0.00008 - 0.006)
		Site 2		
G[c]	4	103	0.561	0.00136
H (no gloves)	4	71.9	4.108	0.01428
I (no gloves)	4	63.8	3.001	0.01176
J	4	85.1	0.831	0.00244
K	4	61.5	0.921	0.00374
L	4	74.2	1.152	0.00388
	Geometric mean (95% C.I.):			0.0045 (0.001 - 0.02)
		Site 3		
M[c]	5.6	93.2	1.143	0.00219
N	5.6	90.5	2.006	0.00396
O	5.6	71.9	1.039	0.00258
P	5.6	71.9	0.745	0.00185
Q	5.6	91.9	0.647	0.00126
R	5.6	105	0.207	0.00035
	Geometric mean (95% C.I.):			0.0016 (0.0004-0.006)
		All Sites Combined		
	Geometric mean (95% C.I.):			0.004 (0.0003 - 0.014)

[a] Middendorf (1992), Table 2, pp. 7-8
[b] Middendorf (1992), Table 3, p. 25
[c] Mixer
C.I. = Confidence Interval

Table 3-5: Estimated absorption rates for workers involved in backpack applications of triclopyr BEE as Garlon 4 from the study by Spencer et al. (2000).

Worker	Amount Handled (lb a.e.)[a]	Body Weight (kg)[b]	Amount Excreted in Urine (mg)[c]	Exposure Rate (mg/kg bw·lb a.e.)
		Day 1 (7/10/95)		
1	3.12	85	5.75	0.0217
2	3	75	1.96	0.0087
3	3.12	63.6	3.53	0.0178
4	3.37	77.3	3.12	0.0120
5	3.37	79.5	3.57	0.0133
6	3.37	75	1.12	0.0044
7	3.25	61.4	0.81	0.0041
8	3.25	75	9.45	0.0388
9	3.5	72.7	4.12	0.0162
10	3.25	58.2	2.86	0.0151
		Geometric mean (95% C.I.):		0.0158 (0.004 - 0.0388)
		Day 2 (7/11/95)		
1	3.67	85	6.16	0.0197
2	2.66	75	3.89	0.0195
3	3.67	63.6	8.81	0.0377
4	2.91	77.3	3.81	0.0169
5	2.91	79.5	2.49	0.0108
6	2.91	75	1.57	0.0072
7	3.42	61.4	2.70	0.0129
8	3.54	75	11.05	0.0416
9	3.16	72.7	2.65	0.0115
10	3.16	58.2	4.66	0.0253
		Geometric mean (95% C.I.):		0.0206 (0.0071 - 0.0442)

[a] Table VI in Spencer et al. 2000, p. 21
[b] Table IV in Spencer et al. 2000, p. 17
[c] Table IX in Spencer et al. 2000, p. 25

Table 3-6: Summary of Exposure Scenarios for the General Public at an Application Rate of 1 lb/acre

Scenario	Target	Dose (mg/kg/day)			Worksheet
		Central	Lower	Upper	
Acute/Accidental Exposures					
Direct spray, entire body	Child	2.42e-01	5.32e-02	3.50e+00	Acid D01a
		6.99e-01	2.94e-03	5.72e+00	BEE D01a
Direct spray, lower legs	Woman	2.43e-02	5.34e-03	3.51e-01	Acid D01b
		7.03e-02	2.95e-04	5.75e-01	BEE D01b
Dermal, contaminated vegetation	Woman	3.00e-02	1.17e-02	6.59e-02	Acid D02
		6.59e-02	6.80e-04	8.47e-02	BEE D02
Contaminated fruit [1]	Woman	3.11e-03	3.11e-03	3.45e-02	Both D03
Contaminated water, spill	Child	2.73e-01	1.04e-01	2.05e+00	Both D05
Contaminated water, stream	Child	6.77e-03	4.59e-05	4.51e-02	Both D06
Consumption of fish, general public	Man	4.92e-04	3.08e-04	2.46e-03	Both D08a
Consumption of fish, subsistence populations	Man	2.40e-03	1.50e-03	1.20e-02	Both D08b
Chronic/Longer Term Exposures					
Contaminated fruit	Woman	1.52e-03	1.17e-03	2.27e-02	Both D04
Consumption of water	Man	8.57e-04	1.60e-04	1.71e-03	Both D07
Consumption of fish, general public	Man	2.57e-07	6.86e-08	4.29e-07	Both D09a
Consumption of fish, subsistence populations	Man	2.08e-06	5.55e-07	3.47e-06	Both D09b

[1] Because the application is fixed at 1 lb a.e./acre and the lower ranges of residue and consumption rates are the same as the central estimate, the lower range of exposure is equal to the central estimate of exposure for this scenario.

Table 3-7: Chemical specific parameters used in GLEAMS modeling and estimation of concentrations in ambient water.

Parameter	Value	Comment/ Reference
Triclopyr Acid		
Halftimes (days)		
Aquatic Sediment	365	Ritter and Peakcock 2000Wolt 1997a, anaerobic
Foliar	15	Knisel et al. 1992
Soil	46	Knisel et al. 1992
Water	196	Wolt 1997a
Ko/c	20	Diaz-Diaz and Loague 2001
Water Solubility, mg/L	435	USDA/ARS 1995
Foliar wash-off fraction	0.95	
Chlorpyrifos		
Halftimes (days)		
Aquatic Sediment	30.5	USDA/ARS 1995, aerobic
Foliar	3.3	Knisel et al. 1992
Soil	30	Knisel et al. 1992
Water	29	USDA/ARS 1995 ke of 0.0236
Ko/c	9930	USDA/ARS 1995, ranges of Ko/c
Water Solubility, mg/L	0.4	Knisel et al. 1992
Foliar wash-off fraction	0.65	Knisel et al. 1992
TCP		
Halftimes (days)		
Aquatic Sediment	730	No anaerobic value found. By analogy to soil halftime, assume twice the value for triclopyr.
Foliar	4.5	Houtman et al. 1997a, range of 4.0-4.7 for residues in plants
Soil	69.3	Wolt 1997a
Water	6	Houtman et al. 1997a, range of 4.2-7.9.
Ko/c	84	Wolt 1997a, central value
Water Solubility, mg/L	0.1	Estimated from Kow and by analogy to trichlorophenol

Table 3-8: Estimated concentrations of triclopyr in a small stream (4,420 m^3/day) adjacent to a 10 acre plot based on GLEAMS modeling with different soil types and annual rainfall rates and using a normalized application rate of 1 lb triclopyr/acre.

Annual Rainfall	Concentrations in Ambient Water (µg/L per lb/acre)					
	Clay		Loam		Sand	
	Average	Maximum	Average	Maximum	Average	Maximum
5.	0.00000	0.00000	0.00000	0.00000	0.00000	0.00000
10.	0.00008	0.01851	0.03235	5.94422	0.03964	7.69605
15.	0.08969	16.56788	0.11003	18.38273	0.15460	16.74569
20.	0.16379	32.38329	0.19622	31.32508	0.27777	24.39023
25.	0.23425	48.93094	0.27462	43.57765	0.37851	30.90842
50.	0.49493	125.35022	0.53554	93.98586	0.65232	54.75161
100.	0.80275	244.10622	0.79948	168.68464	0.85999	87.83543
150.	0.99332	316.94874	0.95520	222.13533	0.96323	114.52715
200.	1.13863	377.40395	1.07237	268.16636	1.03575	138.84111
250.	1.25534	428.03592	1.16804	308.68609	1.09395	161.48882

Table 3-9: Estimated concentrations of triclopyr in a small pond (0.25 acre, 1 meter deep) adjacent to a 10 acre plot based on GLEAMS modeling with different soil types and annual rainfall rates and using a normalized application rate of 1 lb triclopyr/acre.

Annual Rainfall	Concentrations in Ambient Water (µg/L per lb/acre)					
	Clay		Loam		Sand	
	Average	Maximum	Average	Maximum	Average	Maximum
5.	0.00000	0.00000	0.00000	0.00000	0.00000	0.00000
10.	0.02042	0.04316	8.62360	15.93867	10.51033	18.58442
15.	22.41765	43.69961	26.93346	48.78870	37.62965	60.80427
20.	35.21956	77.93913	43.35316	81.90297	62.02870	112.31133
25.	7.43983	51.05207	37.35390	74.62374	75.04630	155.76596
50.	2.68761	53.16233	3.33549	46.84445	5.23193	32.94249
100.	1.78954	49.06172	1.85062	38.79637	2.31057	25.23622
150.	1.42045	41.61058	1.37904	32.51705	1.52656	20.59004
200.	1.21049	36.75169	1.13495	28.58410	1.16026	17.78559
250.	1.06753	33.34363	0.98174	25.79227	0.94963	15.90318

Table 3-10: Estimated concentrations of TCP in a small stream (4,420 m^3/day) adjacent to a 10 acre plot based on GLEAMS modeling with different soil types and annual rainfall rates and using a normalized application rate of 1 lb triclopyr/acre.

Annual Rainfall	Concentrations in Ambient Water (µg/L per lb/acre)					
	Clay		Loam		Sand	
	Average	Maximum	Average	Maximum	Average	Maximum
5.	0.00000	0.00000	0.00000	0.00000	0.00000	0.00000
10.	0.00020	0.08146	0.01805	0.39835	0.02809	0.69038
15.	0.02777	0.82043	0.05472	1.53444	0.11953	2.78657
20.	0.04616	1.32487	0.08956	2.18105	0.20505	4.27456
25.	0.06262	1.68497	0.11997	2.70293	0.26784	5.18803
50.	0.11179	2.55477	0.20831	3.67571	0.39552	7.29741
100.	0.13958	2.22602	0.25809	4.94882	0.45850	9.30172
150.	0.14969	3.55566	0.27087	5.23047	0.47860	10.16934
200.	0.15020	4.42799	0.27227	5.22180	0.48706	10.67511
250.	0.14750	4.82155	0.26947	5.24954	0.49070	11.01527

Table 3-11: Estimated concentrations of TCP in a small pond (0.25 acre, 1 meter deep) adjacent to a 10 acre plot based on GLEAMS modeling with different soil types and annual rainfall rates and using a normalized application rate of 1 lb triclopyr/acre.

Annual Rainfall	Concentrations in Ambient Water (µg/L per lb/acre)					
	Clay		Loam		Sand	
	Average	Maximum	Average	Maximum	Average	Maximum
5.	0.00000	0.00000	0.00000	0.00000	0.00000	0.00000
10.	0.00178	0.06946	0.16286	0.48679	0.25441	0.81625
15.	0.23296	0.88998	0.44794	1.48595	0.98970	2.68828
20.	0.32997	1.18650	0.64979	1.79595	1.52245	3.54769
25.	0.07938	1.06097	0.55877	1.56085	1.74002	3.77038
50.	0.03499	0.62863	0.06302	0.81529	0.14345	1.35499
100.	0.03184	0.39088	0.04399	0.59434	0.07330	0.67943
150.	0.03707	0.42654	0.04424	0.50324	0.06129	0.44145
200.	0.03958	0.42679	0.04455	0.42679	0.05637	0.42679
250.	0.04142	0.46400	0.04519	0.46400	0.05419	0.46400

Table 3-12: Estimated concentrations of TCP in a small stream (4,420 m^3/day) adjacent to a 10 acre plot based on GLEAMS modelling with different soil types and annual rainfall rates and using a normalized application rate of 1 lb triclopyr/acre and 1 lb chlorpyrifos/acre.

Annual Rainfall	Concentrations in Ambient Water (µg/L per lb/acre)					
	Clay		Loam		Sand	
	Average	Maximum	Average	Maximum	Average	Maximum
5.	0	0	0	0	0	0
10.	0.0011	0.46856	0.04864	0.9487	0.07977	1.58987
15.	0.09563	2.34934	0.15842	3.42725	0.26471	5.53369
20.	0.18268	3.94499	0.28485	5.92321	0.45241	9.37183
25.	0.27946	6.39801	0.4053	8.3665	0.61001	11.87562
50.	0.65245	19.30059	0.8412	21.90566	1.03408	26.86142
100.	0.95801	47.53443	1.15926	52.03928	1.1862	49.4293
150.	0.98183	71.0441	1.15489	82.21434	1.13961	57.54736
200.	0.93551	87.77298	1.06889	113.2465	1.06946	60.56932
250.	0.86697	105.4742	0.97212	110.7397	0.9986	66.7199

Table 3-13: Estimated concentrations of TCP in a small pond (0.25 acre, 1 meter deep) adjacent to a 10 acre plot based on GLEAMS modelling with different soil types and annual rainfall rates and using a normalized application rate of 1 lb triclopyr/acre and 1 lb chlorpyrifos/acre.

Annual Rainfall	Concentrations in Ambient Water (µg/L per lb/acre)					
	Clay		Loam		Sand	
	Average	Maximum	Average	Maximum	Average	Maximum
5.	0	0	0	0	0	0
10.	0.01004	0.39912	0.43871	1.16637	0.72254	1.93844
15.	0.79554	2.61144	1.29629	3.50984	2.19362	5.68347
20.	1.28145	3.66607	2.06263	5.00968	3.35924	8.0809
25.	0.37508	4.07917	1.94484	5.91705	3.98105	9.22834
50.	0.19497	4.3966	0.28846	5.4175	0.44852	7.27958
100.	0.16165	4.72915	0.20459	6.00989	0.23187	5.7612
150.	0.14494	4.72386	0.17099	6.16144	0.1709	4.20375
200.	0.13207	4.6083	0.14698	6.17536	0.14308	3.5753
250.	0.12326	4.44299	0.13066	4.87113	0.12832	3.20886

Table 3-14: Summary of risk characterization for workers based on an application rate of 1 lb/acre and the chronic RfD of 0.05 mg/kg/day and the acute RfD of 1 mg/kg/day.

Scenario	Hazard Quotient		
	Central	Lower	Upper
General Exposures Chronic RfD			
Directed ground spray (Backpack)	3e-01	9e-03	1.6
Broadcast ground spray (Boom spray)	4e-01	1e-02	3
Aerial applications	3e-01	5e-03	1.6
General Exposures Acute RfD			
Directed ground spray (Backpack)	1e-02	5e-04	8e-02
Broadcast ground spray (Boom spray)	2e-02	7e-04	2e-01
Aerial applications	1e-02	2e-04	8e-02
TEA - Accidental/Incidental Exposures, Acute RfD			
Immersion of Hands, 1 minute	2e-05	7e-06	3e-04
Contaminated Gloves, 1 hour	1e-03	4e-04	2e-02
Spill on hands, 1 hour	6e-03	1e-03	9e-02
Spill on lower legs, 1 hour	2e-02	3e-03	2e-01
BEE - Accidental/Incidental Exposures, Acute RfD			
Immersion of Hands, 1 minute	8e-03	3e-03	7e-02
Contaminated Gloves, 1 hour	5e-01	2e-01	4
Spill on hands, 1 hour	2e-02	8e-05	2e-01
Spill on lower legs, 1 hour	5e-02	2e-04	4e-01

[1] See Table 3-2 for a summary of the exposures used in calculating the hazard quotients.

Table 3-15: Summary of risk characterization for the general public at an application rate of 1 lb/acre.

Scenario	Target	Hazard Quotient			Formulation
		Central	Lower	Upper	
Acute/Accidental Exposures					
Direct spray, entire body	Child	2e-01	5e-02	3	Acid
		7e-01	3e-03	6	BEE
Direct spray, lower legs	Woman	5e-01	1e-01	7	Acid
		1.4	6e-03	11	BEE
Dermal, contaminated vegetation	Woman	6e-01	2e-01	1.3	Acid
		1.3	1e-02	1.7	BEE
Contaminated fruit [2]	Woman	6e-02	6e-02	7e-01	Both
Contaminated water, spill	Child	3e-01	1e-01	2.0	Both
Contaminated water, stream	Child	7e-03	5e-05	5e-02	Both
Consumption of fish, general public	Man	5e-04	3e-04	2e-03	Both
Consumption of fish, subsistence populations	Man	2e-03	1e-03	1e-02	Both
Chronic/Longer Term Exposures					
Contaminated fruit	Woman	3e-02	2e-02	5e-01	Both
Consumption of water	Man	2e-02	3e-03	3e-02	Both
Consumption of fish, general public	Man	5e-06	1e-06	9e-06	Both
Consumption of fish, subsistence populations	Man	4e-05	1e-05	7e-05	Both

[1] See Table 3-6 for a summary of the exposures used in calculating the hazard quotients.
[2] Because the application is fixed at 1 lb a.e./acre and the lower ranges of residue and consumption rates are the same as the central estimate, the lower range of exposure is equal to the central estimate of exposure for this scenario.

4. ECOLOGICAL RISK ASSESSMENT

4.1. HAZARD IDENTIFICATION

4.1.1. Overview. An assessment of the potential toxic hazards associated with the exposures of wildlife mammalian species to triclopyr is based on the same studies on experimental mammals that are used in the human health risk assessment. Although triclopyr causes developmental effects only at doses that cause maternal toxicity, reproductive effects are obviously an endpoint of concern to both the human health and ecological risk assessments and the quantitative risk assessment for mammalian wildlife is based on the same data as used in the human health risk assessment. For birds, the most relevant data for this risk assessment are the standard dietary and bird reproduction studies required for registration as well as the acute oral LD_{50} studies. The acute oral LD_{50} values of triclopyr range from 849 mg/kg to 2055 mg/kg, similar to the range seen in experimental mammals. Several subchronic dietary studies have been conducted on triclopyr acid, triclopyr TEA, and triclopyr BEE (Garlon 4). Based on these studies, the U.S. EPA/OPP (1998a) has classified triclopyr acid as being practically non-toxic to slightly toxic to birds and triclopyr TEA and triclopyr BEE (Garlon 4) as practically non-toxic to birds. As in experimental mammals, triclopyr has also been tested for reproductive effects in birds. The LOAEL for reproductive toxicity in birds is 500 ppm in the diet or about 50 mg/kg bw with a corresponding NOAEL of 20 ppm in the diet or about 20 mg/kg bw. These values are marginally higher than the NOAEL of 5 mg/kg bw and LOAEL of 25 mg/kg bw in mammals. Based on standard bioassays in the honey bee, U.S. EPA has classified triclopyr as practically non-toxic to bees. No additional studies on the toxicity of triclopyr or triclopyr formulations to terrestrial invertebrates have been encountered. Little information is available on the toxicity of triclopyr to terrestrial microorganisms. Very high concentrations of triclopyr has been shown to cause growth inhibition in bacteria and fungi in laboratory bioassays.

Triclopyr mimics indole auxin plant growth hormones and cause uncontrolled growth in plants. The U.S. EPA requires studies of seedling emergence and vegetative vigor in non-target plants for herbicides. Triclopyr BEE is about equally toxic in both types of assays with the lowest NOEC being 0.0036 lb/acre for seeding emergence and 0.0039 lb/acre for vegetative vigor. Triclopyr TEA, on the other hand, is much less toxic in the seedling emergence assay, with a NOEC of 0.333 lb/acre. For the most sensitive species tested, the NOEC for triclopyr TEA in the vegetative vigor assay is 0.0041 lb/acre, about the same as that of triclopyr BEE. The least sensitive species, however, had a much higher NOEC of 0.0111 lb/acre. As field study indicates that some bryophytes and lichens may be sensitive to long term effects after triclopyr exposure.

In addition to the laboratory bioassays and field observations on single species or related groups of species, there are a number field studies that have assessed the effects of triclopyr on terrestrial organisms, both animal and plant. There is very little suggestion in any of the field studies that triclopyr had any direct adverse effect on terrestrial species and most reported effects may simply reflect changes in habitat secondary to vegetation management practices.

As with terrestrial species, the acute lethal potency of triclopyr and triclopyr formulations has been relatively well-defined. There is a major difference in the potential hazards posed by triclopyr TEA formulations (e.g., Garlon 3A) and triclopyr BEE formulations (e.g., Garlon 4) to fish but there are no remarkable differences among species in terms of sensitivity to the various agents covered in this risk assessment. The sublethal effects of Garlon 4 on a salmonid (rainbow trout) has been assayed: at concentrations of 0.32-0.43 mg/L, about a factor of 2 below the 96-hour LC_{50}, fish were lethargic. At levels $\leq$0.1 mg/L, fish were hypersensitive over 4-day periods of exposure. This is reasonably consistent with the threshold for behavioral changes in rainbow trout for Garlon 4 of 0.6 mg/L. The corresponding threshold for behavioral changes to Garlon 3A was 200 mg/L is consistent with the relative acute lethal potencies of these two agents. Subchronic toxicity data are available only on the triethylamine salt of triclopyr and only in fathead minnows. The survival of fathead minnows (embryo-larval stages) was significantly reduced at 253 mg/L compared with control animals. At 162 mg/L, there was a slight decrease in body length.

The observation of hind limb deformities in free-living amphibians has substantially increased concern for the effects of xenobiotics on populations of amphibians. Garlon 3A and Garlon 4 have been specifically tested for malformations in the frog embryo teratogenesis assay and no statistically significant effects were noted. In studies on embryos and tadpoles of three species of frogs using Garlon 4, exposures to 0.6, 1.2, and 4.6 ppm a.e. caused no effect on hatching success, malformations, or subsequent avoidance behavior of embryos but the two higher concentrations were associated with mortality or immobility in tadpoles.

Based on acute lethality, aquatic invertebrates appear to be about equally or somewhat less sensitive than fish to the various forms of triclopyr. The only chronic toxicity data involves a reproduction study in daphids in which the NOEC was 80.7 mg/L with a corresponding LOEC of 149 mg/L.

Based on EC_{50} values, triclopyr TEA is about equally toxic to both algae (lowest EC_{50} of 5.9 ppm a.i.) and macrophytes (lowest EC_{50} of 8.8 ppm a.i.). As with toxicity to fish and invertebrates, triclopyr BEE is more toxic with EC_{50} values as low as 0.88 ppm a.i. for macrophytes and 0.1 ppm for algae. Efficacy studies are available on the use of Garlon 3A to control unwanted aquatic vegetation. At levels of 0.25-2.5 mg a.e./L (as Garlon 3A) over time periods of 2-48 hours, very little effect was seen for exposure periods less than 6 hours. At 0.25 mg/L, effective control was associated with exposure periods of 24 (partially effective) to 72 (very effective) hours.

TCP (an environmental metabolite of tryclopyr) is substantially more toxic in fish than either triclopyr acid or triclopyr TEA, with acute LC_{50} values in the range of about 2 to 10 ppm, similar to the toxicity of triclopyr BEE. An early life-stage study has been conducted with TCP in rainbow trout yielding an NOEC of 0.0808 mg/L and an LOEC of 0.134 mg/L based on the most sensitive endpoint. Thus, TCP appears to be much more toxic than triclopyr TEA, for which the

corresponding values in an early life stage study in the fathead minnow are 104 mg/L and 162 mg/L.

4.1.2. Toxicity to Terrestrial Animals.

4.1.2.1. Mammals – As summarized in the human health risk assessment (Section 3.1), there are several standard toxicity studies in experimental mammals that were conducted as part of the registration process as well as some published studies on the toxicity of triclopyr to mammals. Just as these studies are used in the human health risk assessment to identify the potential toxic hazards associated with exposures to triclopyr, they can also be used to identify potential toxic effects in wildlife mammalian species. As summarized in Section 3.1.2, the kidney appears to be the primary target tissue for triclopyr in experimental mammals. In the absence of data on most wildlife species, it seems reasonable to assume that the kidney will also be primary site of action in mammalian wildlife. As detailed in Section 3.1.9, there are a large number of toxicity studies on the reproductive effects of triclopyr. Although triclopyr causes developmental effects only at doses that cause maternal toxicity, reproductive effects are obviously an endpoint of concern to both the human health and ecological risk assessments and the quantitative risk assessment for mammalian wildlife (Section 4.3) will be based on the same data as used in the human health risk assessment.

4.1.2.2. Birds – Information of the toxicity of triclopyr to birds is summarized in Appendix 9. The most relevant data for this risk assessment are the standard dietary and bird reproduction studies required for registration as well as the acute oral LD_{50} studies. As summarized in Appendix 9, the acute LD_{50} values of triclopyr on gavage administration to birds ranges from 849 mg/kg to 2055 mg/kg. This is very similar to the range of 600 to 1000 mg/kg seen in experimental mammals (Section 3.1.3). Based on these data, the U.S. EPA/OPP (1998a) has classified triclopyr as being slightly toxic to birds.

As also summarized in Appendix 9, several subchronic dietary studies have been conducted on triclopyr acid, triclopyr TEA, and triclopyr BEE (Garlon 4). Based on these studies, the U.S. EPA/OPP (1998a) has classified triclopyr acid as being practically non-toxic to slightly toxic to birds and triclopyr TEA and triclopyr BEE (Garlon 4) as practically non-toxic to birds. While information is not available on food consumption rates from these studies, it is likely that the birds consumed about 10% of their body weight in food per day by analogy to the reported food consumption rates in Beavers et. al. (1979, 1980). Thus, the acute dietary LC_{50} values of about 3,000 to 10,000 ppm summarized in Appendix 9 correspond to daily dose of about 300 to 1000 mg/kg bw, reasonably close to the reported gavage LD_{50} values.

As in experimental mammals, triclopyr has also been tested for reproductive effects (Beavers et. al. 1979 and 1980; Mayes 1990a). The LOAEL for reproductive toxicity in birds is 500 ppm in the diet or about 50 mg/kg bw with a corresponding NOAEL of 20 ppm in the diet or about 20 mg/kg bw (Beavers et. al. 1980). These values are marginally higher than the NOAEL of 5 mg/kg bw and LOAEL of 25 mg/kg bw in mammals (Table 3-1), suggesting that birds may not

be any more sensitive than mammals for effects on reproductive function. As detailed in Section 4.3, these studies form the basis of the chronic dose-response assessment for birds.

4.1.2.3. Invertebrates – The honey bee is the standard test organism for assessing the potential effects of pesticides on terrestrial invertebrates. Acute contact toxicity studies in honey bees are available on triclopyr acid and triclopyr TEA (U.S. EPA/OPP 1998a). In both bioassays, the LD_{50} values were over 100 µg/bee and based on these results, U.S. EPA/OPP (1998a) has classified triclopyr as practically non-toxic to bees. No additional studies on the toxicity of triclopyr or triclopyr formulations to terrestrial invertebrates have been encountered.

4.1.2.4. Microorganisms – Little information is available on the toxicity of triclopyr to terrestrial microorganisms. Estok et al. (1989) examined the effects of Garlon 4 at concentrations of 1, 10, 100, 1000, 5000, or 10,000 ppm a.i. in growth medium (agar) over 26-48 day growth periods on three species of fungi (*Cenococcum geophilum, Pisolithus tinctorius,* and *Hebeloma longicaudum*). The results indicate a significant reduction of radial growth in each species at concentrations $\geq$1000 ppm. Total growth inhibition was observed at $\geq$5000 ppm. *Cenococcum geophilum*, the slowest growing fungus, was least sensitive to the effects of triclopyr. In a similar study, Chakravarty and Sidhu (1987) studied the inhibitory effects of triclopyr (specified only as a Garlon formulation with 48% a.i.) over a 30 day growth period in five fungal species: *Hebeloma crustuliniforme, Laccaria laccata, Thelophora americana, Thelophora terrestris*, and *Suillus tomentosus*. The most sensitive species was *Thelophora americana* for which a slight growth inhibition (93.75% of controls) based on dry weight was reported to be statistically significant at 0.1 ppm. In other species, statistically significant decreases in growth were reported between 1 ppm and 10 ppm.

4.1.2.5. Terrestrial Plants – Triclopyr and other pyridinecarboxylic acid herbicides such as picloram mimic indole auxin plant growth hormones and cause uncontrolled growth in plants. These herbicides behave similarly to the chlorophenoxy acid herbicides such as 2,4-D. At sufficiently high levels of exposure, the abnormal growth is so severe that vital functions cannot be maintained and the plant dies (Bovey and Meyer 1981; Coffman et al. 1993; Extoxnet 1996; Hatterman-Valenti et al. 1995).

Acute toxicity studies in non-target plants are summarized in Appendix 10. The U.S. EPA requires studies of seedling emergence and vegetative vigor in non-target plants for herbicides and these studies are summarized in U.S. EPA/OPP (1998a) for both triclopyr TEA and triclopyr BEE. Seedling emergence studies involve application of the test compound to soil containing seedlings. Vegetative vigor studies involves direct foliar applications to young plants. For both types of studies, exposures are expressed in the same units as application rate – i.e., lb a.i./acre. Triclopyr BEE is about equally toxic in both types of assays with the lowest NOEC being 0.0036 lb/acre for seeding emergence and 0.0039 lb/acre for vegetative vigor. Triclopyr TEA, on the other hand, is much less toxic in the seedling emergence assay, with a NOEC of 0.333 lb/acre. For the most sensitive species tested (sunflowers), the NOEC for triclopyr TEA in the vegetative

vigor assay is 0.0041 lb/acre, about the same as that of triclopyr BEE. The least sensitive species (onions), however, had a much higher NOEC of 0.0111 lb/acre.

The higher toxicity of triclopyr BEE in the seedling emergence assay may relate to the more rapid absorption of the BEE form relative to the TEA form. This difference has been demonstrated quantitatively in chickweed, wheat, and barley (Lewer and Owen 1990), and is likely to be true for most other plant species. Variations in species sensitivity to triclopyr BEE appear to be related directly to the rate of metabolic ester hydrolysis by the plant (Lewer and Owen 1990). As with 2,4-D and 2,4,5-T, arid conditions do not affect the rate of triclopyr absorption but do inhibit translocation and thus efficacy (Bollig et al. 1995; Seiler et al. 1993).

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

4.1.2.6. Field Studies – In addition to the laboratory bioassays or field observations on single species, there are a number field studies that have assessed the effects of triclopyr on groups of terrestrial organisms, both animal and plant (Appendix 11).

Many of the field studies summarized in Appendix 11 suggest either no effect or beneficial effects (e.g., Boggs et al. 1991a; Schulz et al. 1992; Schulz and Leslie 1992). Other studies are difficult to interpret as beneficial or detrimental and may simply reflect changes in habitat. For example, Boggs et al. (1991b) noted an increase in the prevalence of bot fly larval infestations in small mammals after triclopyr applications with prescribed burnings. This effect was tentatively attributed to higher soil temperatures during burning and may have nothing to do with the specific use of triclopyr.

There is very little suggestion in any of the field studies that triclopyr had any direct adverse effect on terrestrial species. The study by Leslie et al. (1996) does indicate that white-tailed deer will avoid areas treated with herbicides followed by prescribed burning. There is no indication, however, that this avoidance is associated with a toxic effect from the herbicide. At sufficiently high applications rates damage to some non-target vegetation has been noted (King and Radosevich 1985; Snipes et al. 1991; Street et al. 1992). These effects, however, are to be expected based on the known phytotoxicity of triclopyr (Section 4.1.2.5).

A concern with the direct use of field studies in a risk assessment is that field studies, like many epidemiology studies, may be difficult to interpret because of the nature of the "control group" and because some studies may not be sufficiently sensitive to detect subtle adverse effects. Nonetheless, some field studies may be useful in conjunction with more controlled laboratory studies in helping to clarify or expand the risk characterization. This is particularly true for potential reproductive effects, which are a potential concern for triclopyr. At the level used for field applications of triclopyr, however, no adverse effects have been noted on reproductive activity in mammals (McMurry et al. 1993a,b; McMurry et al. 1994).

4.1.3. Toxicity to Aquatic Organisms.

4.1.3.1. Fish – As with terrestrial species, the acute lethal potency of triclopyr and triclopyr formulations has been relatively well-defined. These values are typically expressed as time-specific LC_x values where *x* is the estimate of the proportion of fish that die – e.g., 96 hour LC_{50}. A large number of acute LC_{50} values have been determined in various species of fish. These are summarized in Appendix 12.

There is a major difference in the potential hazards posed by triclopyr TEA formulations (e.g., Garlon 3A) and triclopyr BEE formulations (e.g., Garlon 4) to fish. The most extensive comparative study on the toxicity of triclopyr TEA and triclopyr BEE was conducted by Wan et al. (1989). This publication summarizes a series of static bioassays on several species of salmonids that were conducted over a 4-month period in 1986 and a 2-month period in 1987. The 96-hour LC_{50} values for triclopyr acid, triclopyr BEE, Garlon 3A, and Garlon 4 are summarized in Table 4-1. This table also presents the expected LC_{50} values for Garlon 3A and Garlon 4 based on the concentrations and toxicities of triclopyr acid and triclopyr BEE, respectively, in these formations. Wan et al. (1989) also present LC_{50} values at 24, 38, 72, and 96 hours. Since no strong time/response relationship is apparent, the shorter term results are not discussed further.

There are no remarkable differences among species in terms of sensitivity to the various agents covered in this risk assessment. Wan et al. (1989) do not provide confidence intervals on the LC_{50} values; however, given that the acute bioassays were conducted at different times over a prolonged period and the differences in LC_{50} values among species are relatively slight, this lack of information does not represent a significant data gap. Nonetheless, there is a substantial difference between the toxicity of triclopyr acid and the toxicity of triclopyr BEE, and the difference is reflected in the toxicities of the Garlon formulations. As indicated in Table 4-1, triclopyr BEE is more toxic than triclopyr acid, in terms of acid equivalents, by factors ranging from approximately 10 (rainbow trout, 1÷0.1) to 30 (chum salmon, 1÷0.03).

The results of Wan et al (1989) appear to be expressed in terms of the formulation. The expected LC_{50} values for these formulations, given in the fourth column of Table 4-1, are simply the reported LC_{50} values for the active agent divided by the proportion of the agent in the formulation (see footnote in Table 4-1 for details). Garlon 4 is more toxic than Garlon 3A by a factor of about 200 (150-230). This difference in toxicity is substantially greater than the difference in

toxicity between triclopyr BEE and triclopyr acid. As indicated in the last column of Table 4-1, this increased difference appears to be attributable to the less than expected toxicity of Garlon 3A, based on the level of triclopyr acid in this formulation. The level of triclopyr BEE in Garlon 4 appears to account for practically all of the toxicity of Garlon 4 (i.e., the ratios of observed to predicted LC_{50} values do not vary remarkably from unity for Garlon 4). Although Garlon 4 contains kerosene (see section 2.2), the toxicity of kerosene to aquatic species is approximately 100-1,000 fold less than triclopyr BEE [LC_{50} values of approximately 200-3,000 mg/L (CHEMBANK 1995)], supporting the observation that the toxicity of Garlon 4 can be completely accounted for by the toxicity of triclopyr BEE.

The Wan et al. (1989) study is supported by more recent flow-through toxicity assays on Garlon 4 with reported LC_{50} values for salmonids of 0.79-1.76 mg/L (Kreutzweiser et al. 1994) and 0.84 mg/L (Johansen and Geen 1990). As indicated in Appendix 6, Kreutzweiser et al. (1994) report a strong time-response relationship between exposure periods of 1-24 hours. This is not inconsistent with the results of Wan et al. (1989) but simply indicates that increasing body burdens occur during the first 24 hours of exposure.

The sublethal effects of Garlon 4 on salmonid (rainbow trout) has been examined by Johansen and Geen (1990) using flow-through systems. At concentrations of 0.32-0.43 mg/L, about a factor of 2 below the 96-hour LC_{50} determined by these investigators, fish were lethargic. At levels $\leq$0.1 mg/L, fish were hypersensitive over 4-day periods of exposure. This is reasonably consistent with the threshold for behavioral changes in rainbow trout for Garlon 4 of 0.6 mg/L (Morgan et al. 1991). The corresponding threshold for behavioral changes to Garlon 3A was 200 mg/L (Morgan et al. 1991) is consistent with the relative acute lethal potencies of these two agents.

Subchronic toxicity data are available only on the triethylamine salt of triclopyr and only in fathead minnows (Mayes et al. 1984; Mayes 1990c). In this study, fathead minnow eggs were exposed to concentrations of 26, 43, 65, 104, 162, and 253 mg/L for 28 days covering the development from egg to fry. The survival of fathead minnows (embryo-larval stages) was significantly reduced at 253 mg/L compared with control animals. At 162 mg/L, there was a slight decrease in body length. No effects were noted at any of the lower concentrations.

4.1.3.2. Amphibians – The observation of hind limb deformities in free-living amphibians has substantially increased concern for the effects of xenobiotics on populations of amphibians (e.g., Quellet et al. 1997).

Garlon 3A and Garlon 4 have been specifically tested for malformations in the frog embryo teratogenesis assay (Perkins et al. 2000). In this assay, frog (*Xenopus laevis*) embryos are exposed to the test solution in petri dishes for 96-hours. As in the fish bioassays, Garlon 3A was much less toxic than Garlon 4. The LC_5 and LC_{50} values for Garlon 3A were 3,779 and 5,407 mg a.e./L respectively. The corresponding values for Garlon 4 were 6.7 and 9.3 mg/L respectively. No hind limb abnormalities were reported in this study. The only abnormalities

specified in the publication include uncoiling of the gut, edema, blistering, abnormal pigmentation, and axial twisting in control embryos. No statistically significant increase in abnormalities were seen in any groups exposed to Garlon 3A or Garlon 4 at levels that were not lethal. The precise number and nature of abnormalities in the groups exposed to lethal concentrations of the triclopyr formulations are, however, not specified. Nonetheless, this report is consistent with the much large body of studies on reproductive toxicity in mammals (Section 3.1.9) indicating that the triclopyr formulations are not likely to cause reproductive or teratogenic effects at sublethal concentrations.

Berrill et al. (1994) also assayed the toxicity of Garlon 4 in using embryos and tadpoles of *Rana pipiens* (leopard frog), *Rana clamitans* (green frog), and *Rana catesbeiana* (bullfrog) in a static assay with aeration that was conducted a 15° in darkness to prevent hydrolysis of triclopyr BEE. Exposures to 0.6, 1.2, and 4.6 ppm (triclopyr a.e.) caused no effect on hatching success, malformations, or subsequent avoidance behavior of embryos. Newly hatched tadpoles died or became immobile after exposure to the two higher concentrations. The approximate EC_{50} for response to prodding was between 1.2 and 4.6 ppm after a 24 hour exposure period.

4.1.3.3. Aquatic Invertebrates – Information regarding the toxicity to aquatic invertebrates of various forms of triclopyr as well as the commercial formulations are presented in Appendix 13. The available LC_{50} values, while not as extensive as those for fish, suggest that most invertebrates are about equally or somewhat less sensitive than fish to the various forms of triclopyr. Some families of invertebrates (Ephemeroptera, Plecoptera, Trichoptera, Odonata) are much more resistant than fish to Garlon 4 (Kreutzweiser et al. 1992) (Appendix 7).

Only one chronic study on aquatic invertebrates has been encountered. This is a standard daphnid reproduction study that was submitted to U.S. EPA (MRID 00151959, Gersich 1982) and published in the open literature (Gersich et al., 1984). *Daphnia magna* adults were exposed to concentrations of 80.7, 149, 290, 574, and 1177 mg/L for 21 days. At 80.7 mg/L, no significant effects were noted on mean number of broods, total young produced, mean number of young per brood or mean size of young. At the next higher concentration, there was a statistically significant decrease in total young produced, mean number of young per brood. The U.S. EPA/OPP (1998a) used the NOEC of 80.7 mg/L and LOEC of 149 mg/L in their risk assessment of triclopyr.

4.1.3.4. Aquatic Plants – Information regarding the toxicity to aquatic plants of various forms of triclopyr as well as the commercial formulations are presented in Appendix 14. Triclopyr and triclopyr formulations have been subject to a standard set of bioassays in aquatic plats, both algae and macrophytes, that are required for the registration of herbicides. Based on EC_{50} values, triclopyr TEA is about equally toxic to both algae (lowest EC_{50} of 5.9 ppm a.i.) and macrophytes (lowest EC_{50} of 8.8 ppm a.i.). As with toxicity to fish and invertebrates, triclopyr BEE is more toxic with EC_{50} values as low as 0.88 ppm a.i. for macrophytes and 0.1 ppm for algae. A published study by Peterson et al. (1994) reports inhibition of carbon fixation at 2.6 ppm

triclopyr acid. Gardener et al. (1997) reports a NOEC of 0.08 ppm for the effect of Garlon 3A on cell density in a green alga, *Ankistrodesmus* sp.

In addition to the standard toxicity bioassays summarized in Appendix 14, Peterson et al. (1994) examined the effects of triclopyr on carbon fixation in several algal species. The investigators noted no or relatively little inhibition at concentrations of triclopyr acid of 2.6 mg/L.

Efficacy studies are available on the use of Garlon 3A to control unwanted aquatic vegetation. For example, Netherland and Getsinger (1992) examined the effect of Garlon 3A on eurasian watermilfoil, an aquatic macrophyte. At levels of 0.25-2.5 mg a.e./L (as Garlon 3A) over time periods of 2-48 hours, very little effect was seen for exposure periods less than 6 hours. At 0.25 mg/L, effective control was associated with exposure periods of 24 (partially effective) to 72 (very effective) hours (Netherland and Getsinger 1992).

4.1.3.5. Toxicity of TCP to Aquatic Organisms – As summarized in Section 3.1.15, TCP (3,5,6-trichloro-2-pyridinol) is a major metabolite of triclopyr and is found in both soil and water. In mammals, TCP has about the same toxicity as triclopyr. As summarized in Appendix 15, TCP is substantially more toxic in fish than either triclopyr acid or triclopyr TEA, with acute LC_{50} values in the range of about 2 to 10 ppm, similar to the toxicity of triclopyr BEE.
In addition to the acute toxicity studies, an early life-stage study has been conducted in rainbow trout (Marino et al. 1999). The most sensitive endpoint involved growth – i.e., length and weight – with an NOEC of 0.0808 mg/L and an LOEC of 0.134 mg/L. Thus, TCP appears to be much more toxic than triclopyr TEA, for which the corresponding values in an early life stage study in the fathead minnow are 104 mg/L and 162 mg/L (Appendix 12). The TCP study was required by the U.S. EPA/OPP (1998a) but completed after the RED was published. This study impacts the aquatic risk assessment for triclopyr is considered further in the dose-response assessment for fish (Section 4.3.3.1).

4.2. EXPOSURE ASSESSMENT

4.2.1. Overview. Terrestrial animals might be exposed to any applied herbicide from direct spray, the ingestion of contaminated media (vegetation, prey species, or water), grooming activities, or indirect contact with contaminated vegetation. The highest exposures for terrestrial vertebrates will occur after the consumption of contaminated vegetation or contaminated insects. In acute exposure scenarios, doses as high as 112 mg/kg are estimated. Other routes of exposure, like the consumption of contaminated water or direct spray, lead to lower levels of exposure. In chronic exposure scenarios, the higher estimated daily doses are in the range of about 1 to 29 mg/kg/day and are associated with highly conservative assumptions regarding the consumption of contaminated vegetation.

The primary hazards to non-target terrestrial plants are associated with unintended direct deposition or spray drift. Unintended direct spray will result in an exposure level equivalent to the application rate. At least some plants that are sprayed directly with triclopyr at or near the recommended range of application rates will be damaged. Based on the AgDRIFT model, no more than 0.0058 of the application rate would be expected to drift 100 m offsite after low boom ground applications. In order to encompass a wide range of field conditions, GLEAMS simulations were conducted for clay, loam, and sand at annual rainfall rates from 5 to 250 inches. Under arid conditions (i.e., annual rainfall of about 10 inches or less), there is no or very little runoff. Under these conditions, degradation, not dispersion, accounts for the decrease of triclopyr concentrations in soil. At higher rainfall rates, plausible offsite movement of triclopyr results in runoff losses that range from about negligible up to about 0.4 of the application rate, depending primarily on the amount of rainfall rather than differences in soil type, with somewhat greater runoff predicted for triclopyr TEA compared to triclopyr BEE.

For triclopyr TEA, the potential for effects on aquatic species are based on estimated concentrations of triclopyr in water that are identical to those used in the human health risk assessment without additional elaboration. The maximum concentrations of triclopyr in water from the direct application of Garlon 3A for the control of submerged weeds will be similar to the lower to central estimates of concentrations of triclopyr in water after an accidental spill of Garlon 3A. An elaboration of the exposure assessment for triclopyr BEE is, however, required because there are substantial differences in the toxicity of triclopyr TEA and triclopyr BEE to aquatic species and substantial differences in the environmental fate of triclopyr TEA and triclopyr BEE. For this risk assessment, a separate set of GLEAMS models were made using triclopyr BEE as the parent compound and triclopyr acid as the metabolite.

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

In this exposure assessment, estimates of oral exposure are expressed in the same units as the available toxicity data (i.e., oral LD_{50} and similar values). 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 body weight. For dermal exposure, the units of measure usually are expressed in mg of agent per cm^2 of surface area of the organism and abbreviated as mg/cm^2. In estimating dose, however, a distinction is made between the exposure dose and the absorbed dose. The *exposure dose* is the amount of material on the organism (i.e., the product of the residue level in mg/cm^2 and the amount of surface area exposed), which can be expressed either as mg/organism or mg/kg body weight. The *absorbed dose* is the proportion of the exposure dose that is actually taken in or absorbed by the animal.

For the exposure assessments discussed below, general allometric relationships are used to model exposure. In the biological sciences, allometry is the study of the relationship of body size or mass to various anatomical, physiological, or pharmacological parameters (e.g., Boxenbaum and D'Souza 1990). Allometric relationships take the general form:

$$y = aW^x$$

where W is the weight of the animal, y is the variable to be estimated, and the model parameters are **a** and **x**. For most allometric relationships used in this exposure assessment, **x** ranges from approximately 0.65 to 0.75. These relationships dictate that, for a fixed level of exposure (e.g., levels of a chemical in food or water), small animals will receive a higher dose, in terms of mg/kg body weight, than large animals. Thus, estimates of exposure are given for both a small and a large mammal as well as a small and a large bird.

The exposure assessments for terrestrial animals are summarized in Tables 4-2 and 4-3 for triclopyr TEA and triclopyr BEE formulations, respectively. As with the human health exposure assessment, the computational details for each exposure assessment presented in this section are provided in the worksheets (worksheets F01 through F14) of Supplement 1 (triclopyr acid) and Supplement 2 (triclopyr BEE). The scenarios and exposure assessment methods are identical for each formulation and consequently several of the exposure values are identical in Tables 4-2 and 4-3. Two different tables are presented because all exposure scenarios involving estimates of dermal exposure rates differ for triclopyr TEA and triclopyr BEE formulations because of differences in estimated dermal absorption rates. In addition, as discussed in Section 4.3, some of the toxicity values used in the risk characterization differ between triclopyr TEA and triclopyr BEE. Consequently, the use of two exposure tables for each type of formulation simplifies the presentation of the risk characterization (Section 4.4).

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

For this risk assessment, three groups of direct spray exposure assessments are conducted. The first, which is defined in worksheet F01, involves a 20 g mammal that is sprayed directly over

one half of the body surface as the chemical is being applied. The range of application rates as well as the typical application rate is used to define the amount deposited on the organism. The absorbed dose over the first day (i.e., a 24-hour period) is estimated using the assumption of first-order dermal absorption. In the absence of any data regarding dermal absorption in a small mammal, the estimated absorption rate for humans is used (see section 3.1.3). An empirical relationship between body weight and surface area (Boxenbaum and D'Souza 1990) is used to estimate the surface area of the animal. The estimates of absorbed doses in this scenario may bracket plausible levels of exposure for small mammals based on uncertainties in the dermal absorption rate of triclopyr and triclopyr BEE.

Other, perhaps more substantial, uncertainties affect the estimates for absorbed dose. For example, the estimate based on first-order dermal absorption does not consider fugitive losses from the surface of the animal and may overestimate the absorbed dose. Conversely, some animals, particularly birds and mammals, groom frequently, and grooming may contribute to the total absorbed dose by direct ingestion of the compound residing on fur or feathers. Furthermore, other vertebrates, particularly amphibians, may have skin that is far more permeable than the skin of most mammals.

Quantitative methods for considering the effects of grooming or increased dermal permeability are not available. As a conservative upper limit, the second exposure scenario, detailed in worksheet F02a, is developed in which complete absorption over day 1 of exposure is assumed.

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

Direct spray scenarios are not given for large mammals. As noted above, allometric relationships dictate that large mammals will be exposed to lesser amounts of a compound in any direct spray scenario than smaller mammals.

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

Unlike the human health risk assessment in which transfer rates for humans are available, there are no transfer rates available for wildlife species. As discussed in Durkin et al. (1995), the transfer rates for humans are based on brief (e.g., 0.5- to 1-hour) exposures that measure the

transfer from contaminated soil to uncontaminated skin. Species of wildlife are likely to spend longer periods of time, compared to humans, in contact with contaminated vegetation.

It is reasonable to assume that for prolonged exposures a steady-state may be reached between levels on the skin, rates of absorption, and levels on contaminated vegetation, although there are no data regarding the kinetics of such a process. The bioconcentration data on triclopyr (Section 3.2.3.5) as well as its high water solubility and low octanol/water partition coefficient suggest that triclopyr is not likely to partition from the surface of contaminated vegetation to the surface of skin, feathers, or fur. Thus, a plausible but conservative partition coefficient is unity (i.e., the concentration of the chemical on the surface of the animal will be equal to the dislodgeable residue on the vegetation).

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

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

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

A large herbivorous mammal is included because empirical relationships of concentrations of pesticides in vegetation, discussed below, indicate that grasses may have substantially higher pesticide residues than other types of vegetation such as forage crops or fruits (Worksheet A04). Grasses are an important part of the diet for some large herbivores, but most small mammals do

not consume grasses as a substantial proportion of their diet. Thus, even though using residues from grass to model exposure for a small mammal is the most conservative approach, it is not generally applicable to the assessment of potential adverse effects. Hence, in the exposure scenarios for large mammals, the consumption of contaminated range grass is modeled for a 70 kg herbivore, such as a deer. Caloric requirements for herbivores and the caloric content of vegetation are used to estimate food consumption based on data from U.S. EPA/ORD (1993). Details of these exposure scenarios are given in worksheets F10 for acute exposures as well as Worksheets F11a and F11b for longer-term exposures.

For the acute exposures, the assumption is made that the vegetation is sprayed directly – i.e., the animal grazes on site – and that the animal consumes 100% of a contaminated diet. While appropriately conservative for acute exposures, neither of these assumptions are plausible for longer-term exposures. Thus, for the longer-term exposure scenarios for the large mammal, two sub-scenarios are given. The first is an on-site scenario that assumes that a 70 kg herbivore consumes short grass for a 90 day period after application of the chemical. The contaminated vegetation accounts for 10 to 100% of the diet assuming that the animal would spend 10 to 100% of the grazing time at the application site. Because the animal is assumed to be feeding at the application site, drift is set to unity - i.e., direct spray. This scenario is detailed in Worksheet 12a. The second sub-scenario is similar except the assumption is made that the animal is grazing at distances of 25 to 100 feet from the application site (lowering risk) but that the animal consumes 100% of the diet from the contaminated area (increasing risk). For this scenario, detailed in Worksheet F12b, AgDRIFT is used to estimate deposition on the off-site vegetation. Drift estimates from AgDrift are summarized in Worksheet A06 and this model is discussed further in Section 4.2.3.2.

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

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

Similarly, the consumption of contaminated insects is modeled for a small (10g) bird. No monitoring data have been encountered on the concentrations of triclopyr in insects after applications of triclopyr. The empirical relationships recommended by Fletcher et al. (1994) are used as surrogates as detailed in Worksheet F14. To be conservative, the residue rates from small insects are used – i.e., 45 to 135 ppm per lb/ac – rather than the residue rates from large insects – i.e., 7 to 15 ppm per lb/ac.

As indicated in Section 3.2.3.6, the empirical relationships recommended by Fletcher et al. (1994) for fruit appear to substantially overestimate concentrations on fruit based on the study by Siltanen et al. (1981). Thus, the data from Siltanen et al. (1981) are used for all exposure scenarios involving contaminated fruit. A detailed discussion of the basis for this approach is given in Section 3.2.3.6.

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

4.2.2.4. Soil Microorganisms – As discussed in Section 4.1.2.4, the available data on the toxicity of triclopyr to soil microorganisms is based on studies in which exposure is characterized as concentrations of triclopyr in soil or growth media. Thus, for the exposure assessment of soil microorganisms, comparable exposures are required.

Initial concentrations of triclopyr or any other compound in soil surface can be calculated simply as a function of application rate and depth of incorporation. For example, an application rate of 1 lb/acre is equivalent to 11.21 $\mu g/cm^2$. Assuming a shallow depth of incorporation of 1 cm, the corresponding concentration in soil would be 11.21 $\mu g/cm^3$ or about 11.21 ppm using a soil density of unity. At an application rate of 10 lbs/acre, the highest application rate considered in this risk assessment, the initial concentration in the top 1 cm of soil can be similarly calculated as 112.1 ppm.

While these high initial concentrations are considered in the risk characterization (Section 4.4), they represent only a brief period of exposure and will not reflect to dissipation and degradation of triclopyr over time and in deep soil layers. Consequently, longer term concentrations in soil were modeled using GLEAMS as discussed in Section 3.2.3.4.1 and detailed in Attachment 3. Results of the GLEAMS modeling of soil concentrations in the top 1 foot of soil are summarized in Table 4-4 for an application rate of 1 lb/acre. The use of shallower or deeper soil depths would result in greater or less concentrations.

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

4.2.3.1. Direct Spray – Unintended direct spray will result in an exposure level equivalent to the application rate. For many types of herbicide applications - e.g., rights-of-way management, it is plausible that some non-target plants immediately adjacent to the application site could be

sprayed directly. This type of scenario is modeled in the human health risk assessment for the consumption of contaminated vegetation.

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

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

Estimates of drift for ground applications is given in Worksheet A06. In ground broadcast applications, triclopyr will typically be applied by low boom ground spray and thus these estimates are used in the current risk assessment. Drift associated with backpack (directed foliar applications) are likely to be much less. This is discussed further in the risk characterization.

Drift distance can be estimated using Stoke's law, which describes the viscous drag on a moving sphere. According to Stoke's law:

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

or

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

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

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

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

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

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

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

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

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

4.2.3.3. Runoff – Triclopyr or any other herbicide may be transported to off-site soil by runoff or percolation. Both runoff and percolation are considered in estimating contamination of ambient water. For assessing off-site soil contamination, however, only runoff is considered. The approach is reasonable because off-site runoff will contaminate the off-site soil surface and could impact non-target plants. Percolation, on the other hand, represents the amount of the herbicide that is transported below the root zone and thus may impact water quality but should not affect off-site vegetation. Based on the results of the GLEAMS modeling (Section 3.2.3.4.2), the proportion of the applied triclopyr was estimated for clay, loam, and sand at rainfall rates ranging from 5 inches to 250 inches per year. These results are summarized in Worksheet G04.

4.2.3.4. Wind Erosion – Wind erosion is a major transport mechanism for soil (e.g., Winegardner 1996) and is associated with the environmental transport of herbicides (Buser 1990). Although numerous models were developed for wind erosion (e.g., Strek and Spaan 1997, Strek and Stein 1997), the quantitative aspects of soil erosion by wind are extremely complex and site specific. Field studies conducted on agricultural sites found that annual wind erosion may account for soil losses ranging from 2 to 6.5 metric tons/ha (Allen and Fryrear 1977). The upper range reported by Allen and Fryrear (1977) is nearly the same as the rate of 2.2 tons/acre (5.4 tons/ha) recently reported by the USDA (1998). The temporal sequence of soil loss (i.e., the amount lost after a specific storm event involving high winds) depends heavily on soil characteristics as well as meteorological and topographical conditions.

This risk assessment uses average soil losses ranging from 1 to 10 tons/ha·year, with a typical value of 5 tons/ha·year. The value of 5 tons/ha·year is equivalent to 500 g/m^2 [1 ton=1000 kg and 1 ha = 10,000 m^2] or 0.05 g/cm^2 [1m^2=10,000 cm^2]. Thus, using a soil bulk density of 1.5 g/cm^3 (Knisel et al. 1992, p. 56), the depth of soil removed from the surface per year would be 0.033 cm[(0.05 g/cm^2)÷ (1.5 g/cm^3)]. The average amount per day would be about 0.00009 cm/day [0.033 cm per year ÷ 365 days/year]. The upper range of the typical daily loss would thus be about 0.00018 cm/day.

The amount of triclopyr that might be transported by wind erosion depends on several factors, including the application, the depth of incorporation into the soil, the persistence in the soil, the wind speed, and the topographical and surface conditions of the soil. Under desirable conditions, like relatively deep (10 cm) soil incorporation, low wind speed, and surface conditions that inhibit wind erosion, it is likely that wind transport of triclopyr would be neither substantial or nor significant.

Any number of undesirable exposure scenarios could be constructed. As a reasonable 'worst case' scenario, it is assumed that triclopyr is applied to arid soil, that it is incorporated into the top 1 cm of soil, that minimal rainfall occurs for a 2-month period, that the degradation and dispersion of triclopyr in the soil is negligible over the 2-month period, and that local conditions favor a high rate of soil loss (i.e., smooth, sandy surface with high wind speeds) that is a factor at the central estimate of the typical rate (i.e., 0.00009 cm/day). Under those conditions, 0.0054 [0.00009 cm/day × 60 days ÷ 1 cm] of the applied triclopyr would be lost due to wind erosion. This is virtually identical to the estimates of off-site contamination from low-boom applications at a distance of 100 feet from the application site and is greater than drift that would be expected 500 feet offsite (0.0016 for low-boom applications from Worksheet A06) by a factor about 3 [0.0054 ÷ 0.0016 = 3.375]. Thus, in areas where wind erosion of soil may occur, wind erosion could be a more important mode of offsite movement than drift during application.

The deposition of the triclopyr contaminated soil also will vary substantially with local conditions. Under desirable conditions, the soil might be dispersed over a very large area and be of no toxicological consequence. In some cases, however, local topographical conditions might favor the deposition and concentration of contaminated dust from a large treated area into a relatively small off-site area. An objective approach for modeling these types of events is not available and, for this risk assessment, neither concentration nor dispersion is considered quantitatively.

4.2.4. Aquatic Organisms. For triclopyr TEA, the potential for effects on aquatic species are based on estimated concentrations of triclopyr in water that are identical to those used in the human health risk assessment (Section 3.2.3.4) without additional elaboration. As noted in Section 3.2.3.4.4, the maximum concentrations of triclopyr in water from the direct application of Garlon 3A for the control of submerged weeds will be similar to the lower to central estimates of concentrations of triclopyr in water after an accidental spill of Garlon 3A. This is discussed further in the risk characterization for aquatic organisms (Section 4.4.3.1).

An elaboration of the exposure assessment for triclopyr BEE is, however, required. As detailed further in 4.3.3, there are substantial differences in the toxicity of triclopyr TEA and triclopyr BEE to aquatic species. There are also substantial differences, however, in the environmental fate of triclopyr TEA and triclopyr BEE. Both of these factors much be considered in the risk assessment.

As reviewed by U.S. EPA (1998a), triclopyr TEA will dissociate almost instantaneously to triclopyr acid in water. Thus, the toxicity of triclopyr TEA and triclopyr acid are essentially the same when expressed as acid equivalents. Triclopyr BEE, on the other hand, will degrade quickly but not instantaneously to triclopyr acid. This makes a substantial difference in the results from acute toxicity bioassays because, as summarized in Tables 2-1 and 2-2, the octanol water partition coefficient for triclopyr BEE (about 10,233) is higher than that of triclopyr acid (about 0.35 at pH 7) by a factor of nearly 30,000 [10,233÷0.35=29,237]. The much higher octanol water partition coefficient for triclopyr BEE will lead to much more rapid uptake of this form relative to triclopyr acid and this probably accounts for the much higher acute toxicity of triclopyr BEE relative to triclopyr acid.

For this risk assessment, a separate set of GLEAMS models were made using triclopyr BEE as the parent compound and triclopyr acid as the metabolite. The specific parameters used for triclopyr BEE are summarized in Table 4-5. The parameters used for triclopyr acid were identical to those used in the human health risk assessment and are given in Table 3-7. Most of the model parameters for triclopyr BEE are taken directly from Knisel et al. (1999) or U.S. EPA (1998a). The only exception is the halftime for triclopyr BEE in aquatic sediments. No values for this parameter were encountered in the literature and for the modeling the halftime was set at the halftime for hydrolysis. Since the triclopyr BEE to triclopyr acid is very rapid and since water is a substantial component of aquatic sediment, this approach may provide a reasonable approximation.

The results of the GLEAMS modeling for maximum concentrations of triclopyr BEE in a small stream are given in Table 4-6. For sand, the maximum concentration of triclopyr BEE (149 ppb) is very similar to that of triclopyr acid (161 ppb from Table 3-8) because both forms of triclopyr will rapidly leach in very sandy soils after heavy rainfall. Since the maximum concentrations from the GLEAMS modeling is based on a rainfall event that occurs one day after application, relatively little triclopyr BEE is transformed to triclopyr acid and the peak concentrations are essentially equivalent. For both clay and loam soils, the maximum concentrations of triclopyr BEE (66 ppb in clay and 92 ppb in loam) are less than that of triclopyr acid (428 ppb for clay and 308 ppb for loam from Table 3-8) because of the somewhat higher binding to organic matter in soil and consequent lesser runoff of triclopyr BEE relative to triclopyr acid in these soils.

Table 4-6 does not include average values and tables for concentrations in lakes, similar to Table 3-9 in the human health risk assessments, are not provided. While these model runs were made, this risk assessment will adopt the approach taken by U.S. EPA/OPP (1998a) in the chronic aquatic risk assessment for triclopyr – i.e., because triclopyr BEE is rapidly degraded to triclopyr

acid, all chronic risks are based on concentrations of triclopyr acid rather than triclopyr BEE. Thus, concentrations of triclopyr BEE from Table 4-6 are used on Worksheet G03 for the characterization of acute risks to aquatic species. The upper range of triclopyr BEE concentrations is taken at 150 ppb, the approximate concentration associated with runoff from sandy soil at an annual rainfall rate of 250 inches per year. The central estimate is taken as 0.014 mg/L per lb applied per acre. As in the human health risk assessment, this is the geometric mean of the range and the approximate value of maximum concentrations in stream water modeled for all soils at an annual rainfall rate of 50 inches per year. The lower range of the maximum concentration is set at 0.0003 mg/L, the lowest concentration greater than zero from Table 4-6. Lower concentrations are of course plausible but this has no impact on the characterization of risk.

4.3. DOSE-RESPONSE ASSESSMENT

4.3.1. Overview. The dose-response assessment for terrestrial mammals is based on the same toxicity values that form the basis of the RfDs used in the human health risk assessment: an acute NOAEL of 100 mg/kg/day and a chronic NOAEL of 5 mg/kg/day. For birds, the acute NOAEL is taken as 535 mg/kg/day for triclopyr acid and 388 mg/kg/day for triclopyr BEE. These based on the 5-day dietary concentrations of 5357 ppm acid equivalents and 3884 ppm acid equivalents for triclopyr TEA and triclopyr BEE. For chronic exposures, the NOAEL is taken as 10 mg/kg/day for both forms of triclopyr. Because triclopyr BEE is rapidly converted to triclopyr acid, chronic exposures to triclopyr BEE are implausible. The only information on the toxicity of triclopyr to terrestrial invertebrates is the standard studies in honey bees that are required for pesticide registration that report an LD_{50} values were over 100 µg/bee. Laboratory studies involving responses in artificial growth media suggest that responses in soil microorganisms may be highly variable among species with growth unaffected in some species at concentrations of up to 1000 ppm in growth medium but inhibited in other species are concentrations as low as 0.1 ppm. The applicability of these studies to assessing the risk to soil microorganisms from exposures to triclopyr in soil is questionable but these are the only data available.

For terrestrial plants, the risk characterization for triclopyr will be based on the standard assays used by U.S. EPA for both triclopyr TEA and triclopyr BEE. For vegetative vigor, the most sensitive NOAEL for triclopyr TEA is 0.0041 lb/acre and the corresponding value for triclopyr BEE is 0.0039 lb/acre and both of these values are used directly for the risk characterization. For triclopyr TEA, the risk characterization for effects on seedling emergence from runoff will be based on the NOAEL of 0.333 lb/acre. The corresponding value for triclopyr BEE will be taken as 0.003 lb/acre.

For aquatic species, the U.S. EPA typically uses LC_{50} values or fractions of LC_{50} values as the basis for characterizing risk of acute exposures in fish. In the U.S. EPA/OPP RED on triclopyr, an acute LC_{50}equivalent to 199 ppm a.e. is used to characterize acute risks to freshwater fish for triclopyr TEA and an acute LC_{50} value of 0.25 ppm a.e. is used to characterize acute risks for

freshwater fish for triclopyr BEE. For the quantitative risk characterization, the LC_{50} values selected by U.S. EPA/OPP are maintained in this risk assessment.

Data on subchronic and chronic toxicity to fish is scant. Only one subchronic toxicity is available reporting a NOEC of 104 mg/L for triclopyr TEA. This study is relevant both to triclopyr TEA and triclopyr BEE because of the rapid hydrolysis of triclopyr BEE. Thus, for both triclopyr TEA and triclopyr BEE, the NOAEL of 104 mg/L is used to assess chronic toxicity in fish. There are relatively few studies available on amphibians. Because fish are apparently more sensitive to triclopyr, both TEA and BEE, and because of the more extensive toxicity data available on fish, a separate dose-response assessment for amphibians is not conducted. Aquatic invertebrates appear to be as sensitive to both triclopyr TEA and triclopyr BEE as are fish. For this risk assessment, an LC_{50} values of 132.9 mg/L for triclopyr TEA and 8.55 mg/L for triclopyr BEE will be use to characterize acute risks to aquatic invertebrates. For chronic effects on invertebrate species, a chronic NOEC of 80.7 mg/L from a daphnid reproduction study is used for risk characterization.

4.3.2. Toxicity to Terrestrial Organisms.

4.3.2.1. Mammals– As summarized in the dose-response assessment for the human health risk assessment (Section 3.3.2.), the chronic RfD is based on a NOAEL in experimental mammals (a multi-generation reproduction study in rats) of 5 mg/kg/day with an associated LOAEL of 25 mg/kg/day. These NOAEL and LOAEL values are used directly for the chronic risk assessment of terrestrial mammals. Similarly, for acute exposures, the NOAEL and LOAEL values used for the risk assessment of terrestrial organisms are based on the same values used by the U.S. EPA/OPP (2002) for the acute RfD in humans – i.e., 100 mg/kg/day as the NOAEL and 300 mg/kg/day as the LOAEL.

The application of these NOAEL and LOAEL values to small rodents is clearly appropriate, since the NOAEL and LOAEL values come from a studies in rats. Ecological risk assessments, however, are intended to encompass a wide range of mammalian species, from very small animals such as mice and voles to large mammals such as deer. For many chemicals, systematic differences in species sensitivity are apparent and generally indicate that small animals are less sensitive (i.e., have higher toxicity values) than large animals. For triclopyr, the best study in a large mammal for quantitatively comparing differences in sensitivity is the study by Osweiler (1983 summarized in Appendix 4) in which adult Shetland pony geldings weighing151-203 kg were given gavage doses of triclopyr at 0, 60, and 300 mg/kg/day for 4 days. As in the rodent studies, the dose of 300 mg/kg/day was clearly a LOAEL – i.e., horses evidenced gross signs of toxicity including depression and recumbency as well as kidney damage. At 60 mg/kg/day, no adverse effects were noted. Thus, this study suggests that larger mammals are no more sensitive to triclopyr than smaller mammals, although dogs may be an exception (Section 3.3.2).

4.3.2.2. Birds – Based on available LD_{50} values as well as subchronic dietary studies in birds, the U.S. EPA/OPP (1998a, p. 35) has classified triclopyr as slightly toxic to practically non-toxic to birds. As an index of potential toxicity from acute exposure to triclopyr TEA, the U.S. EPA/OPP

(1998a) uses the 5-day dietary study by Fink(1978, MRID 40346503 summarized in Appendix 9) in which the LC_{50} was 11,622 ppm as triclopyr TEA and 5357 ppm as triclopyr acid equivalents. For triclopyr BEE, U.S. EPA/OPP (1998a) uses the 5-day dietary study by Lynn (1991, MRID 41905501 summarized in Appendix 9) in which the LC_{50} was 5401 ppm as triclopyr BEE and 3884 ppm as triclopyr acid equivalents.

For longer-term effects, U.S. EPA/OPP 1998a uses the dietary NOAEL of 100 ppm. This is based on the 1-generation reproduction study in ducks by Beavers et. al. (1980, MRID 00031250, summarized in Appendix 9) as well as the 1-generation reproduction study in quail by Beavers et al. (1979, MRID 00031251). In both studies, dietary concentrations of 100, 200, or 500 ppm corresponded to daily doses of about 10, 20, and 50 mg/kg bw/day based on measured food consumption – i.e., the birds consumed an amount of food per day equivalent to about 10% of their body weight. In both studies, the 100 ppm dietary exposure was classified as a NOAEL and the 200 ppm dietary exposure was classified as a LOAEL.

The U.S. EPA bases the risk characterization on a comparison of the dietary NOAEL values to expected concentrations in vegetation. In this risk assessment, the same studies and NOAEL values will be used but the comparisons will be based on doses expressed as mg/kg body weight. This approach is taken because the direct use of dietary concentrations from laboratory studies may be under-protective. Laboratory diets generally involve the use of dry food. Dry laboratory chow usually has a higher caloric content than food consumed in the wild, if only because most food consumed in the wild has a high water content. In addition, most reported concentrations of a pesticide in environmental samples are given on a wet (natural) weight rather than a dry (dedicated) weight basis. Consequently, animals tend to eat greater amounts of food in the wild than they do under laboratory conditions (U.S. EPA/ORD 1993). Consequently, for a fixed concentration in food, ingested doses expressed as mg/kg bw/day often will be higher in free living animals than in laboratory animals.

Because of these relationships, Forest Service risk assessments use doses expressed as mg/kg body weight for both the exposure and dose-response assessments. As detailed in the worksheets, information on caloric requirements and caloric values of different foods are used to estimate the amount of a particular food that an animal will use.

Thus, for this risk assessment, the acute NOAEL for birds is taken as 535 mg/kg/day for triclopyr acid and 388 mg/kg/day for triclopyr BEE. These based on the 5-day dietary concentrations of 5357 ppm acid equivalents and 3884 ppm acid equivalents for triclopyr TEA (Fink 1978) and triclopyr BEE (Lynn 1991), summarized above, and using the conversion factor of 10% from the chronic oral studies. For chronic exposures, the NOAEL is taken as 10 mg/kg/day for both forms of triclopyr.

4.3.2.3. Terrestrial Invertebrates – As discussed in Section 4.1.2.3, the only information on the toxicity of triclopyr to terrestrial invertebrates is the standard studies in honey bees that are required for pesticide registration that report an LD_{50} values were over 100 µg/bee. Taking the

LD_{50} of 100 µg/bee and using a body weight of 0.093 g for the honey bee (USDA/APHIS 1993), the 100 µg/bee dose corresponds to about 1075 mg/kg bw [0.100 mg/0.000093 kg = 1075.27 mg/kg]. This value will be used in the risk characterization for assessing effects of direct contact on terrestrial invertebrates. Given the large number of species of terrestrial invertebrates, the use of data from a single species for the risk characterization obviously leads to uncertainty in the risk assessment.

4.3.2.4. Terrestrial Microorganisms – As discussed in Section 4.1.2.4, the studies by Estok et al. (1989) and Chakravarty and Sidhu (1987) suggest that responses in soil microorganisms may be highly variable among species with growth unaffected in some species at concentrations of up to 1000 ppm in growth medium but inhibited in other species are concentrations as low as 0.1 ppm.

The applicability of these studies to assessing the risk to soil microorganisms from exposures to triclopyr in soil is questionable. It is plausible that triclopyr in a growth medium will have a greater bioavailability than triclopyr in soil. Nonetheless, these studies using growth medium exposures are the only data encountered in the conduct of this risk assessment and thus will be applied directly to the estimated concentrations of triclopyr in soil.

4.3.2.5. Terrestrial Plants – The risk characterization for triclopyr will be based on the standard assays used by U.S. EPA/OPP (1998a) for both triclopyr TEA and triclopyr BEE. These are described in Section 4.1.2.5 and summarized in Appendix 10. Separate risk characterizations will be considered for both runoff and drift. The risks associated with runoff are based on seedling emergence assays and the risks associated with drift are based on assays for vegetative vigor.

For vegetative vigor, the most sensitive NOAEL for triclopyr TEA is 0.0041 lb/acre and the corresponding value for triclopyr BEE is 0.0039 lb/acre and both of these values are used directly for the risk characterization.

For triclopyr TEA, the risk characterization for effects on seedling emergence from runoff will be based on the NOAEL of 0.333 lb/acre. The corresponding value for triclopyr BEE will be taken as 0.003 lb/acre. Both of these are identical to the lowest NOEC values cited by U.S. EPA/OPP (1998a). For both triclopyr TEA and triclopyr BEE, the range of values for toxicity to different plant species is relatively narrow and thus a range of values is not used in this risk assessment to reflect differences in species sensitivity.

4.3.3. Aquatic Organisms.

4.3.3.1. Fish and Amphibians – As detailed in Section 4.1.3.1, there are major differences in the potential hazards posed by acute exposures to triclopyr TEA formulations (e.g., Garlon 3A) and triclopyr BEE formulations (e.g., Garlon 4) to fish and these differences are reflect in the U.S. EPA/OPP (1998a) risk assessment as well as the current risk assessment.

The U.S. EPA typically uses LC_{50} values or fractions of LC_{50} values as the basis for characterizing risk of acute exposures in fish. For example, in the U.S. EPA/OPP (1998a) RED on triclopyr uses an acute LC_{50} value of 279 ppm a.i., equivalent to 199 ppm a.e., from an acute bioassay in fathead minnows to characterize risk for freshwater fish for triclopyr TEA (U.S. EPA/OPP 1998a, p. 82). Similarly, for triclopyr BEE, the U.S. EPA/OPP (1998a) RED on triclopyr uses an acute LC_{50} value of 0.36 ppm a.i., equivalent to 0.25 ppm a.e., from an acute bioassay in bluegill sunfish to characterize risk for freshwater fish for triclopyr BEE (U.S. EPA/OPP 1998a, p. 83).

A common concern with this approach is that more subtle non-lethal effects, that may impact of the stability of fish populations in the field, may not be properly assessed. In some respects, this concern is somewhat misguided. Most acute fish toxicity studies, as summarized in Appendix 12, report the results as LC_{50} values and there are sound statistical reasons for this approach (e.g., Finney 1971). In addition, the U.S. EPA/OPP sets a based level of concern for hazard quotients based on LC_{50} values at 0.05 (U.S. EPA/OPP 1998a, p. 71). In other words, if the expected exposure is equal to one-twentieth (0.05) of the LC_{50}, the Agency judges that there may be a cause for concern at least in sensitive or endangered species. This is essentially similar to the use of an uncertainty factor as in the human health risk assessments. In addition, as noted by Janz et al. (1991), sublethal exposures of coho salmon to various formulations of triclopyr do not appear to cause signs of physiological stress.

Thus, for the quantitative risk characterization (Section 4.4), the LC_{50} values selected by U.S. EPA/OPP (1998a) will be maintained in this risk assessment. For the current risk assessment, the approach used to characterize acute risks to fish has very little impact on the interpretation of risk. As discussed further in Section 4.4, the acute risks associated with the use of triclopyr TEA are extremely low but the risks associated with the use of triclopyr BEE are obvious.

While the acute toxicity of triclopyr to fish is characterized relatively well, data on subchronic and chronic toxicity is scant. Only one subchronic toxicity is available (Mayes et al. 1984) and this study covers only on the triclopyr TEA in fathead minnows. At 140 mg/L over an exposure period of 28 days, the survival of fathead minnows (embryo-larval stages) was significantly reduced, compared with control animals and no effects were noted at concentrations of 104 mg/L or less (Mayes et al. 1984; Mayes 1990c). As discussed by U.S. EPA/OPP (1998a), this study is relevant both to triclopyr TEA and triclopyr BEE because triclopyr BEE will rapidly hydrolyze to triclopyr acid in water and "chronic" exposure to triclopyr BEE is implausible. Thus, for both triclopyr TEA and triclopyr BEE, the NOAEL of 104 mg/L is used to assess chronic toxicity in fish.

As discussed in Section 4.1.3.4 and detailed in Appendix 15, TCP is about as acutely toxic to fish as triclopyr BEE. For assessing the acute hazards of exposure to TCP, the lowest acute LC_{50} value is used – i.e., 1.8 ppm in Coho salmon from the study by Wan (1987). For longer term exposures, the early life-stage study in rainbow trout (Marino et al. 1999) is used with a NOAEL of 0.0808 mg/L.

As summarized in Section 4.1.3.2, there are relatively few studies available on amphibians. All toxicity values that might be used for amphibians are substantially higher than those used for fish. Because fish are apparently more sensitive to triclopyr, both TEA and BEE, and because of the more extensive toxicity data available on fish, a separate dose-response assessment for amphibians is not conducted and risks to fish are used to characterize potential risks to amphibians.

4.3.3.2. Aquatic Invertebrates– As indicated in Section 4.1.3.3 and detailed in Appendix 7, aquatic invertebrates appear to be as sensitive to both triclopyr TEA and triclopyr BEE as are fish. For triclopyr acid, the U.S. EPA/OPP (1998a) uses an acute LC_{50} value of 132.9 mg/L, about the same as the 199 ppm a.e. value used for fish. As with fish, aquatic invertebrates are more sensitive to triclopyr BEE. The U.S. EPA/OPP (1998a) uses an acute LC_{50} value of 12 mg/L for triclopyr BEE based on an acute toxicity study in daphnia (MRID00151965, Milazzo 1981). While this risk assessment generally attempts to remain consistent with values selected by the U.S. EPA, the more recent study by Peterson et al. (2001) provides data on several invertebrate species with LC_{50} values ranging from 8.55 mg/L for an *Ameletus* (Mayfly) species to 45 mg/L for *Lepidostoma unicolor*, a species of caddisfly. Although not substantially different than the value use by U.S. EPA (1998a), the somewhat more conservative LC_{50} of 8.55 mg/L will be use to characterize acute risks to aquatic invertebrates.

For chronic effects on invertebrate species, the daphnid reproduction study by Gersich et al. (1984) will be used. As summarized in Section 4.1.3.3, this is the only chronic study available and defines a NOEC of 80.7 mg/L and LOEC of 149 mg/L in their risk assessment of triclopyr. This is also the study used by U.S. EPA/OPP (1998a) in the RED on triclopyr.

4.3.3.3. Aquatic Plants– For triclopyr TEA, the U.S. EPA/OPP (1998a) uses EC_{50} values of 8.8 ppm a.i. for macrophytes and 5.9 ppm a.i. for algae. Since these two values are reasonably close to each other, this risk assessment with characterize risks to aquatic plants based on the lower value, 5.9 ppm a.i. which is equivalent to 4.2 ppm a.e. This value is used in G03 of Supplement 1 for the risk characterization of aquatic plants.

For triclopyr BEE, the U.S. EPA/OPP (1998a) uses EC_{50} values of 0.88 ppm a.i. for macrophytes and 0.1 ppm a.i. for algae. Again, the lower value, equivalent to 0.07 ppm a.e., is used for risk characterization and this value is used in G03 of Supplement 2for the risk characterization of aquatic plants.

4.4. RISK CHARACTERIZATION

4.4.1. Overview. For terrestrial mammals, the central estimates of hazard quotients do not exceed the level of concern for any exposure scenarios. At the upper range of exposures, the hazard quotients exceed the level of concern for large mammals and large birds consuming contaminated vegetation exclusively at the application site.

At higher application rates, concern for exposure scenarios involving the consumption of contaminated vegetation is augmented substantially. At the maximum application rate of 10 lbs a.e./acre, the central estimate of the hazard quotient exceed the level of concern for several acute exposure scenarios: the direct spray of a small mammal assuming 100% absorption, a large mammal consuming contaminated vegetation, and a small bird consuming contaminated insects. The central estimates of the hazard quotients for the chronic consumption of vegetation is exceeded for a large mammal and a large bird and the upper range on the hazard quotients are also increased by a factor of 10: i.e., to 60 for a large mammal and 50 for a large bird. This risk assessment is consistent with the risk characterization given by U.S. EPA indicating that contaminated vegetation is primary concern in the used of triclopyr and that high application rates will exceed the level of concern for both birds and mammals in longer term exposure scenarios.

Some effects may be anticipated on nontarget vegetation under some conditions. Because of the relatively low toxicity of triclopyr TEA compared to triclopyr BEE, the risk characterization for triclopyr TEA is much less severe than that of triclopyr BEE. At an application rate of 1 lb/acre, potentially damaging runoff from triclopyr TEA would be anticipated only at relatively high rainfall rates. While a lesser amount of triclopyr BEE will runoff, the higher toxicity of triclopyr BEE leads to hazard quotients above the level of concern starting are relatively modest rainfall rates – i.e., 20 to 25 inches per year. At an application rate of 10 lbs a.e./acre per acre, damage due to runoff after the application of triclopyr TEA would be expected at annual rainfall rates as low as 20 inches per year. For triclopyr BEE, the hazard quotients are of concern for all but the most arid areas. The potential impact of offsite drift of triclopyr varies substantially with the application rate. At an application rate of 1 lb a.e./acre, potentially damaging exposures could occur within about 100 feet of the application site. At the maximum application rate of 10 lbs a.e./acre, damaging drift could occur at distances of over 1000 feet from the application site.

The risk characterization for aquatic organisms differs for triclopyr TEA and triclopyr BEE. For triclopyr TEA, risks to aquatic species are low over the entire range of application rates that may be used in Forest Service programs. At the highest projected application rate, the hazard quotient for acute risks to aquatic plants from runoff into streams would reach unity. For acute risks to aquatic plants in the application of triclopyr TEA directly to water for the control of submerged weeds, the hazard quotient of 0.6 is based on the targeted water concentration given on the product label.

Although triclopyr BEE is much more toxic to aquatic species than triclopyr TEA or triclopyr

acid, the projected levels of exposure are much less even for acute scenarios because of the rapid hydrolysis of triclopyr BEE to triclopyr acid as well as the lesser runoff of triclopyr BEE because of it's lower water solubility and higher affinity for soils. Nonetheless, triclopyr BEE is projected to be somewhat more hazardous when used near bodies of water where runoff to open water may occur. At an application rate of 1 lb a.e./acre, the level of concern for acute exposure to aquatic plants is exceeded at the upper range of projected concentrations. At an application rate of 10 lbs a.e./acre, the level of concern for acute exposure to aquatic plants is exceeded at the central estimate as well as the upper range of projected concentrations.

The risk characterization for TCP is considered quantitatively only for fish because toxicity data are available only for fish. At the typical application rate of 1 lb a.e./acre, the worst case hazard quotients are below the level of concern. That the maximum application rate of 10 lbs a.e./acre, the hazard quotients would be a factor of 10 higher and the hazard quotient for longer term exposure would be substantial (HQ=9). Thus, if triclopyr is applied at higher rates of exposure in areas where surface water contamination is plausible, site-specific modeling and/or environmental monitoring would be useful to ensure and verify that concentrations TCP do reach harmful concentrations. Concentrations of TCP in surface water after the application of triclopyr at 1 lb a.e./acre and chlorpyrifos at 1 lb a.e./acre are well below a level of concern. Thus, the concern for TCP residues in surface water appears to be associated with high application rates of triclopyr rather than applications triclopyr and chlorpyrifos in the same area.

4.4.2. Terrestrial Organisms

4.4.2.1. Terrestrial Animals – The quantitative risk characterization for terrestrial animals is summarized in Table 4-7 for triclopyr TEA formulations and Table 4-8 for triclopyr BEE formulations. These hazard quotients are calculated by dividing the exposure assessments , summarized in Table 4-2 for triclopyr TEA and Table 4-2 for triclopyr BEE, by the toxicity values specified at the bottom of Tables 4-7 or 4-8. Similar to the risk characterization tables for human health (Table 3-14 and Table 3-15), the hazard quotients are the level of exposure divided by the toxicity value (given at the bottom of Table 4-7 and Table 4-8) then rounded to one or two significant decimal places or digits. Hazard quotients >1 and ≤2 are shown to two significant digits. All others are rounded to one significant decimal place or integer. All hazard quotients that are below the level of concern – i.e., a hazard quotient below unity – are expressed in scientific notation. All hazard quotients greater than unity are expressed in fixed point decimal notation and highlighted with a shaded background.

Tables 4-7 and 4-8 are based on an application rate of 1 lb a.e./acre. As indicated in Section 2, this is the typical application rate in Forest Service programs. At the application rate of 1 lb a.e./acre, no hazard quotients exceed unity for any species based on acute exposure scenarios and no central estimates of the hazard quotients for chronic exposure scenarios exceed unity for longer term exposures.

The acute exposure scenarios for birds are based on an LD_{50} rather than an acute NOAEL. Thus, following the approach taken by U.S. EPA/OPP (1998a, p. 70), the level of concern for birds is

based on a hazard quotient of 0.1 rather than unity. All other hazard quotients are based on a level of concern of unity.

For birds, the acute hazard quotient based on the consumption of contaminated vegetation in the application of triclopyr TEA reaches the level of concern (i.e., a hazard quotient of 0.1) and the corresponding hazard quotient for triclopyr BEE exceeds the level of concern (i.e., a hazard quotient of 0.2) at the upper range of plausible exposures.

Two chronic scenarios, both involving the consumption of contaminated vegetation, exceed unity at the upper range of possible exposure – the large mammal and large bird consuming contaminated vegetation on site. For the large mammal consuming contaminated vegetation at the application site, the hazard quotient of 4 corresponds to a daily dose of 18.2 mg/kg/day. As discussed in Section 4.3.2.1, the chronic NOAEL for a mammal is 5 mg/kg/day with a corresponding LOAEL of 25 mg/kg/day. For the large bird consuming contaminated vegetation at the application site, the hazard quotient of 3 corresponds to a daily dose of 28.6 mg/kg/day. As discussed in Section 4.3.2.2, the chronic NOAEL for a bird is 10 mg/kg/day with a corresponding LOAEL of 20 mg/kg/day. Thus, for the large bird but not the large mammal, the estimated exposure exceeds both the NOAEL and the LOAEL. As with the corresponding risk assessment for human health effects (Section 3.2.3.6) this is an extraordinarily conservative scenario.

At higher application rates, concern for exposure scenarios involving the consumption of contaminated vegetation is augmented substantially. As indicated in Section 2.4, the highest application rate covered by this risk assessment is 10 lb a.e./acre. At this application rate, all of the hazard quotients summarized in Tables 4-7 and 4-8 are increased by a factor 10. Based on this application rate, the central estimate of the hazard quotient exceed the level of concern for several acute exposure scenarios: the direct spray of a small mammal assuming 100% absorption (HQ=2), a large mammal consuming contaminated vegetation (HQ=2), and a small bird consuming contaminated insects (HQ=1, LOC=0.1). The central estimates of the hazard quotients for the chronic consumption of vegetation is exceeded for a large mammal (HQ=5) and a large bird (HQ=4) and the upper range on the hazard quotients are also increased by a factor of 10: i.e., 40 for a large mammal and 30 for a large bird.

While the U.S. EPA/OPP (1998a) uses somewhat different exposure scenarios and application rates, the risk characterization for terrestrial animals given in this risk assessment is consistent with the risk characterization given by U.S. EPA/OPP (1998a) indicating that contaminated vegetation is primary concern in the used of triclopyr and that high application rates will exceed the level of concern for both birds and mammals in longer term exposure scenarios.

4.4.2.2. Terrestrial Plants– The quantitative risk characterizations for terrestrial plants are given for both runoff and drift. The risk characterizations for runoff are given in Tables 4-9 and 4-10 for triclopyr TEA and triclopyr BEE formulations, respectively. As in the risk characterization tables for terrestrial animals, an application rate of 1 lb a.e./acre is used in both Table 4-9 and

Table 4-10. The estimates of runoff are taken from the GLEAMS models used in the human health risk assessment to estimate the contamination of water (Section 3.2.3.4.1). No concentration or dilution of runoff is assumed – i.e., the runoff is assumed to go from a treated acre and be evenly distributed over an untreated acre. The U.S. EPA/OPP (1998a) refers to this as "sheet runoff" and also conducts an exposure assessment for "channelized" runoff in which runoff from a 10 acre plot is concentrated in a one-acre plot. The U.S. EPA/OPP (1998a), however, uses only three categories for estimating runoff based on water solubility: 1 %, 2 %, and 5% for water solubility of <10 ppm, 10-100 ppm, and >100 ppm, respectively. Thus, runoff is estimated as 5% for triclopyr TEA and 1% for triclopyr BEE. For the current risk assessment, the use of the GLEAMS model provides a full use of the available data and considers the impact of different amounts of rainfall. Thus, while the use of the "channelized" runoff assumption by U.S. EPA/OPP (1998a) may be viewed as more conservative, the use of very high rainfall rates with GLEAMS yields higher estimates of runoff that are comparable to the "channelized" runoff assumption use by U.S. EPA/OPP (1998a).

As indicated in Tables 4-9 and 4-10, runoff of triclopyr TEA is expected to be substantially higher than triclopyr BEE and this is consistent with the general assumptions on runoff made by U.S. EPA/OPP (1998a). However, because of the relatively low toxicity of triclopyr TEA in the seed germination assay (NOEC of 0.333 lb/acre) compared to triclopyr BEE (0.003 lb/acre), the risk characterization for triclopyr TEA is much less severe than that of triclopyr BEE. At an application rate of 1 lb/acre, potentially damaging runoff from triclopyr TEA would be anticipated only at relatively high rainfall rates – i.e., 200 inches per year or greater. While a lesser amount of triclopyr BEE will runoff, the higher toxicity of triclopyr BEE leads to hazard quotients above the level of concern starting are relatively modest rainfall rates – i.e., 20 to 25 inches per year.

At an application rate of 10 lbs a.e./acre per acre, the hazard quotients presented in Table 4-9 and Table 4-10 would be increased by a factor of 10. This would alter the risk characterization for triclopyr TEA in that potentially hazardous exposures due to runoff would be expected at annual rainfall rates as low as 20 inches per year. For triclopyr BEE, the hazard quotients are of concern for all but the most arid areas.

The risk characterizations for drift are given in Tables 4-11 and 4-12 for triclopyr TEA and triclopyr BEE formulations, respectively. The risk characterization for drift is based bioassay for vegetative vigor involving direct foliar application. As summarized in Section 4.3.2.5, the NOEC values for triclopyr TEA and triclopyr BEE are similar and thus the risk characterizations are similar. At an application rate of 1 lb a.e./acre, potentially damaging exposures could occur within about 100 feet of the application site. At the maximum application rate of 10 lbs a.e./acre, damaging drift could occur at distances of over 1000 feet from the application site.

4.4.2.3. Microorganisms– The potential for substantial effects on soil microorganisms appears to be low. As summarized in Section 4.3.2.4, experimental studies conducted in artificial growth media suggest a very high degree of variability in the response of soil bacteria and fungi to

triclopyr with NOAELs of up to 1000 ppm in some species and growth inhibition at concentrations as low as 0.1 ppm in other species. As summarized in Table 4-4, an application rate of 1 lb/acre is estimate to result in longer term soil concentrations that are well below 0.1 ppm – i.e., in the range of about 0.02 to 0.05 ppm – and peak concentrations in the range of about 0.2 ppm. Thus, if the laboratory studies are used to characterize risk, transient inhibition in the growth of some bacteria or fungi might be expected. This could result in a shift in the population structure of microbial soil communities but substantial impacts on soil – i.e., gross changes in capacity of soil to support vegetation – do not seem plausible. This is consistent with the field experience in the use of triclopyr to manage vegetation.

4.4.3. Aquatic Organisms. The risk characterization for aquatic species includes the risks associated with triclopyr TEA, triclopyr BEE, and TCP, the major environmental metabolite of triclopyr.

4.4.3.1. Triclopyr – The quantitative risk characterization for triclopyr TEA and triclopyr BEE are summarized in Tables 4-13 and 4-14, respectively. As detailed in Section 4.2.4, the risks of triclopyr BEE to aquatic species are considered uniquely only for acute exposures because triclopyr BEE will rapidly hydrolyze to triclopyr acid. Thus, for chronic exposures, the risks associated with triclopyr BEE are based on projected exposures to and the toxicity of triclopyr acid. As with the risk characterization for terrestrial organisms, the risk characterization tables for aquatic species are based on an application rate of 1 lb a.e./acre. The risk characterization for triclopyr TEA (Table 4-13) includes the direct application of Garlon 3A to standing bodies of water for the control of submerged vegetation – labeled "Acute, Aquatic Weeds" in Table 4-13.

For triclopyr TEA, the risk characterization is reasonably unambiguous. At an application rate of 1 lb/acre, acute and chronic risks to aquatic animals, fish or invertebrates, as well as risk to aquatic plants are low. At the highest application considered in this risk assessment, 10 lbs a.e./acre, the risks to aquatic animals remain substantially below a level of concern.

At the highest projected application rate, the hazard quotient for acute risks to aquatic plants from runoff into streams would reach unity (HQ of 0.1 at 1 lb/acre corresponds to an HQ of 1 at 10 lbs/acre). For acute risks to aquatic plants in the application of Garlon 3A directly to water for the control of submerged weeds, the hazard quotient of 0.6 is based on the targeted water concentration given on the product label (Dow AgroSciences 2002; SePRO 2003a).

For longer term exposures, risks to aquatic plants from the application of triclopyr TEA are substantially below the level of concern even at an application rate of 10 lbs a.e./acre. As detailed in Section 3.2.3.4.2, this risk characterization is based on GLEAMS modeling data and supported by monitoring data and confidence in this risk characterization is high.

Although triclopyr BEE is much more toxic to aquatic species than triclopyr TEA or triclopyr acid, the projected levels of exposure are much less even for acute scenarios. As discussed in Section 4.2.4, this lesser exposure is attributable both to the rapid hydrolysis of triclopyr BEE to

triclopyr acid as well as the lesser runoff of triclopyr BEE because of it's lower water solubility and higher affinity for soils. Nonetheless, triclopyr BEE is projected to be somewhat more hazardous when used near bodies of water where runoff to open water may occur. At an application rate of 1 lb a.e./acre, the level of concern for acute exposure to aquatic plants is exceeded at the upper range of projected concentrations. At an application rate of 10 lbs a.e./acre, the level of concern for acute exposure to aquatic plants is exceeded at the central estimate as well as the upper range of projected concentrations.

4.4.3.2. TCP – The risk characterization for TCP is considered quantitatively only for fish because toxicity data are available only for fish. For applications of triclopyr alone at a rate of 1 lb/acre, the highest peak concentration modeled using GLEAMS is about 0.15 ppm and the highest longer term average concentration is about 0.075 ppm (Table 3-9). Both of these values are associated with runoff in sandy soil into a small pond in a region with an average rainfall rate of 25 inches per year. As indicated in 4.3.3.1, the toxicity values used for fish are 1.8 ppm for acute exposures and 0.0808 mg/L for longer term exposures. Thus, the worst case hazard quotients are a about 0.08 for acute exposures and 0.9 for chronic exposures. These hazard quotients, however, correspond to an application rate of 1 lb a.e./acre. That the maximum application rate of 10 lbs a.e./acre, the hazard quotients would be a factor of 10 higher and the hazard quotient for longer term exposure would be substantial (HQ=9). Thus, if triclopyr is applied at higher rates of exposure in areas where surface water contamination is plausible, site-specific modeling and/or environmental monitoring would be useful to ensure and verify that concentrations TCP do reach harmful concentrations.

Concentrations of TCP in surface water after the application of triclopyr at 1 lb a.e./acre and chlorpyrifos at 1 lb a.e./acre are estimated at peaks of up to 0.1 ppm (Table 3-12) and average concentrations of about 0.004 ppm (Table 3-13). These concentrations are well below levels of concern for acute or chronic exposure – i.e., maximum hazard quotients of about 0.05 for both acute and chronic exposure. At these application rates, chlorpyrifos will increase exposure to TCP but not to concentrations that are anticipated to be toxic. Since chlorpyrifos is not applied in forestry applications at rates higher than 1 lb/acre, no additional modeling of chlorpyrifos was conducted. Thus, the concern for TCP residues in surface water appears to be associated with high application rates of triclopyr rather than applications triclopyr and chlorpyrifos in the same area.

Table 4-1: Acute toxicity of triclopyr and related compounds to various species of salmonids[a].

Test Compound	Species	A: 96-hour LC_{50} values	B: Expected LC_{50} values [b]	A÷B
Garlon 3A	coho salmon	463	26	18
	chum salmon	267	21	13
	sockeye salmon	311	21	15
	rainbow trout	420	21	20
	chinook salmon	275	27	10
Garlon 4	coho salmon	2.1	1.6	1.3
	chum salmon	1.7	0.5	3.4
	sockeye salmon	1.4	0.6	2.3
	rainbow trout	2.7	1.8	1.5
	chinook salmon	2.7	1.8	1.5
	pink salmon	1.2	0.8	1.5
Triclopyr acid (not amine salt)	coho salmon	9.6	N/A	N/A
	chum salmon	7.5		
	sockeye salmon	7.5		
	rainbow trout	7.5		
	chinook salmon	9.7		
	pink salmon	5.3		
Triclopyr BEE	coho salmon	1.0	13	0.08
	chum salmon	0.3	10	0.03
	sockeye salmon	0.4	10	0.04
	rainbow trout	1.1	10	0.1
	chinook salmon	1.1	13	0.08
	pink salmon	0.5	7.4	0.06

[a]Source: Wan et al. (1987). All bioassays conducted at 8-14°C, 10 fish/concentration. Static with aeration. LC_{50} based on measured, rather than, nominal concentrations. Photo-period and lighting conditions not specified.

[b]For Garlon 4, the observed LC_{50} of triclopyr BEE divided by the proportion of Garlon 4, 0.616, which consists of triclopyr BEE. For Garlon 3A, the observed LC_{50} of triclopyr acid divided by the proportion of Garlon 3A, 0.360, which consists of triclopyr acid. For triclopyr BEE, the observed LC_{50} of triclopyr acid divided by the proportion of triclopyr BEE, 0.72, which consists of triclopyr acid.

Table 4-2: Summary of exposure scenarios for terrestrial animals for triclopyr TEA.

Scenario	Dose (mg/kg/day)			Worksheet [a]
	Central	Lower	Upper	
Acute/Accidental Exposures				
Direct spray				
small mammal, first-order absorption	6.92e+00	2.69e+00	1.52e+01	F01
small animal, 100% absorption	2.42e+01	2.42e+01	2.42e+01	F02a
bee, 100% absorption	1.60e+02	1.60e+02	1.60e+02	F02b
Contaminated vegetation				
small mammal	3.30e-01	3.30e-01	4.95e-01	F03
large mammal	1.72e+01	1.72e+01	4.86e+01	F10
large bird	2.69e+01	2.69e+01	7.60e+01	F12
Contaminated water				
small mammal, spill	5.32e-01	3.32e-01	2.66e+00	F05
stream	1.32e-02	1.46e-04	5.86e-02	F06
Contaminated insects				
small bird	3.75e+01	3.75e+01	1.12e+02	F14
Contaminated fish				
predatory bird, spill	3.02e-01	9.42e-02	2.26e+00	F08
Longer-term Exposures				
Contaminated vegetation				
small mammal, on site	1.62e-02	6.20e-03	6.52e-02	F04a
off-site	1.63e-04	3.60e-05	1.22e-03	F04b
large mammal, on site	2.52e+00	6.46e-01	3.20e+01	F11a
off-site	8.49e-02	3.75e-02	5.99e-01	F11b
large bird, on site	3.95e+00	1.01e+00	5.01e+01	F13a
off-site	1.33e-01	5.87e-02	9.37e-01	F13b
Contaminated water				
small mammal	4.39e-03	1.17e-03	7.32e-03	F07
Contaminated fish				
predatory bird	2.49e-03	3.32e-04	6.23e-03	F09

[a] Worksheet numbers refer to Supplement 1

Table 4-3: Summary of exposure scenarios for terrestrial animals for triclopyr BEE.

Scenario	Dose (mg/kg/day)			Worksheet
	Central	Lower	Upper	
Acute/Accidental Exposures				
Direct spray				
small mammal, first-order absorption	1.52e+01	1.57e-01	1.95e+01	F01
small animal, 100% absorption	2.42e+01	2.42e+01	2.42e+01	F02a
bee, 100% absorption	1.60e+02	1.60e+02	1.60e+02	F02b
Contaminated vegetation				
small mammal	3.30e-01	3.30e-01	4.95e-01	F03
large mammal	1.72e+01	1.72e+01	4.86e+01	F10
large bird	2.69e+01	2.69e+01	7.60e+01	F12
Contaminated water				
small mammal, spill	5.32e-01	3.32e-01	2.66e+00	F05
stream	1.32e-02	1.46e-04	5.86e-02	F06
Contaminated insects				
small bird	3.75e+01	3.75e+01	1.12e+02	F14
Contaminated fish				
predatory bird, spill	3.02e-01	9.42e-02	2.26e+00	F08
Longer-term Exposures				
Contaminated vegetation				
small mammal, on site	1.62e-02	6.20e-03	6.52e-02	F04a
off-site	1.63e-04	3.60e-05	1.22e-03	F04b
large mammal, on site	2.52e+00	6.46e-01	3.20e+01	F11a
off-site	8.49e-02	3.75e-02	5.99e-01	F11b
large bird, on site	3.95e+00	1.01e+00	5.01e+01	F13a
off-site	1.33e-01	5.87e-02	9.37e-01	F13b
Contaminated water				
small mammal	4.39e-03	1.17e-03	7.32e-03	F07
Contaminated fish				
predatory bird	2.49e-03	3.32e-04	6.23e-03	F09

Table 4-4: Estimated concentrations of triclopyr in the upper 12 inches of soil based on GLEAMS modeling with different soil types and annual rainfall rates and using a normalized application rate of 1 lb triclopyr/acre.

Annual Rainfall	Concentrations in Soil (ppm per lb/acre)					
	Clay		Loam		Sand	
	Average	Maximum	Average	Maximum	Average	Maximum
5.	0.04851	0.24095	0.04545	0.22585	0.04545	0.22585
10.	0.04825	0.24086	0.04486	0.22447	0.04473	0.22404
15.	0.04665	0.23656	0.04347	0.22144	0.04272	0.22158
20.	0.04513	0.23236	0.04193	0.21832	0.04052	0.21961
25.	0.04368	0.22800	0.04052	0.21542	0.03868	0.21802
50.	0.03830	0.20815	0.03577	0.20378	0.03366	0.21257
100.	0.03196	0.17765	0.03098	0.18685	0.02989	0.20510
150.	0.02805	0.15900	0.02817	0.17478	0.02806	0.19908
200.	0.02507	0.14351	0.02606	0.16439	0.02679	0.19359
250.	0.02267	0.13081	0.02434	0.15524	0.02577	0.18847

Table 4-5: Chemical specific parameters used in GLEAMS modeling and estimation of concentrations in ambient water for triclopyr BEE.

Parameter	Value	Comment/ Reference
Halftimes (days)		
Aquatic Sediment	0.5	Use value for hydrolysis
Foliar	15	Knisel et al. 1999
Soil	0.125	U.S. EPA/OPP 1998a
Water	0.5	U.S. EPA/OPP 1998a
Koc	780	Knisel et al. 1999
Water Solubility, mg/L	7	U.S. EPA/OPP 1998a
Foliar wash-off fraction	0.7	Knisel et al. 1999

Table 4-6: Estimated maximum concentrations of triclopyr BEE in a small stream (4,420 m^3/day) adjacent to a 10 acre plot based on GLEAMS modeling with different soil types and annual rainfall rates and using a normalized application rate of 1 lb a.e. triclopyr BEE /acre.

Annual Rainfall	Concentrations in Ambient Water (µg/L per lb/acre)		
	Clay	Loam	Sane
5.	0.00000	0.00000	0.00000
10.	0.00000	0.26289	1.30545
15.	0.50440	1.01226	3.94213
20.	1.12023	2.12899	7.24575
25.	2.03639	3.42986	10.92676
50.	7.36758	11.33270	31.00311
100.	20.70968	30.21672	74.37735
150.	35.37091	50.47790	118.59386
200.	50.76192	71.40504	140.65991
250.	66.63049	92.77691	149.38375

Table 4-7: Summary of quantitative risk characterization for terrestrial animals for triclopyr TEA at an application rate of 1 lb a.e./acre. [1]

Scenario	Hazard Quotient[2] Central	Lower	Upper
Acute/Accidental Exposures			
Direct spray			
small mammal, first-order absorption	7e-02	3e-02	2e-01
small animal, 100% absorption	2e-01	2e-01	2e-01
bee, 100% absorption	1e-01	1e-01	1e-01
Contaminated vegetation			
small mammal	3e-03	3e-03	5e-03
large mammal	2e-01	2e-01	5e-01
large bird	5e-02	5e-02	1e-01
Contaminated water			
small mammal, spill	5e-03	3e-03	3e-02
small mammal, stream	1e-04	1e-06	6e-04
Contaminated insects			
small bird	7e-02	7e-02	2e-01
Contaminated fish			
predatory bird, spill	6e-04	2e-04	4e-03
Longer-term Exposures			
Contaminated vegetation			
small mammal, on site	3e-03	1e-03	1e-02
off-site	3e-05	7e-06	2e-04
large mammal, on site	5e-01	1e-01	6
off-site	2e-02	7e-03	1e-01
large bird, on site	4e-01	1e-01	5
off-site	1e-02	6e-03	9e-02
Contaminated water			
small mammal	9e-04	2e-04	1e-03
Contaminated fish			
predatory bird	2e-04	3e-05	6e-04

Toxicity Indices [3]

Acute toxicity value for mammal - NOAEL	100	mg/kg
Chronic toxicity value for mammal - NOAEL	5	mg/kg/day
Acute toxicity value for bird -LD_{50}	535	mg/kg
Chronic toxicity value for birds - NOAEL	10	mg/kg/day
Toxicity value for bee -LD_{50} greater than specified value	1075	mg/kg

[1] See Table 4-2 for summary of exposure assessments.
[2] Estimated dose ÷ toxicity index
[3] See Section 4.3 of the risk assessment for a discussion of the dose-response assessments.

Table 4-8: Summary of quantitative risk characterization for terrestrial animals for triclopyr BEE at an application rate of 1 lb a.e./acre [1]

Scenario	Hazard Quotient[2] Central	Lower	Upper
Acute/Accidental Exposures			
Direct spray			
small mammal, first-order absorption	2e-01	2e-03	2e-01
small animal, 100% absorption	2e-01	2e-01	2e-01
bee, 100% absorption	1e-01	1e-01	1e-01
Contaminated vegetation			
small mammal	3e-03	3e-03	5e-03
large mammal	2e-01	2e-01	5e-01
large bird	7e-02	7e-02	2e-01
Contaminated water			
small mammal, spill	5e-03	3e-03	3e-02
small mammal, stream	1e-04	1e-06	6e-04
Contaminated insects			
small bird	1e-01	1e-01	3e-01
Contaminated fish			
predatory bird, spill	8e-04	2e-04	6e-03
Longer-term Exposures			
Contaminated vegetation			
small mammal, on site	3e-03	1e-03	1e-02
off-site	3e-05	7e-06	2e-04
large mammal, on site	5e-01	1e-01	**6**
off-site	2e-02	7e-03	1e-01
large bird, on site	4e-01	1e-01	**5**
off-site	1e-02	6e-03	9e-02
Contaminated water			
small mammal	9e-04	2e-04	1e-03
Contaminated fish			
predatory bird	2e-04	3e-05	6e-04

Toxicity Indices [3]

Acute toxicity value for mammal - NOAEL	100	mg/kg
Chronic toxicity value for mammal - NOAEL	5	mg/kg/day
Acute toxicity value for bird -LD_{50}	388	mg/kg
Chronic toxicity value for birds - NOAEL	10	mg/kg/day
Toxicity value for bee -LD_{50} greater than specified value	1075	mg/kg

[1] See Table 4-3 for summary of exposure assessments.
[2] Estimated dose ÷ toxicity index
[3] See Section 4.3 of the risk assessment for a discussion of the dose-response assessments.

Table 4-9: Summary of Exposure Assessment and Risk Characterization for Terrestrial Plants from Runoff for triclopyr TEA..

Application rate	1	lb/acre	Typical FS rate, Section 2.4.
Sensitive Species (Lowest NOEC)	0.333	lb/acre	Section 4.3.2.4.
Tolerant Species (Highest NOEC)	0.333	lb/acre	Section 4.3.2.4.

Annual Rainfall	Clay	Loam	Sand
	Proportion lost in Runoff		
5	0.00000	0.00000	0.00000
10	3.12e-08	0.00580	0.00750
15	0.01617	0.01790	0.01628
20	0.03160	0.03051	0.02375
25	0.04775	0.04247	0.03013
50	0.12233	0.09169	0.05344
100	0.23824	0.16463	0.08573
150	0.30933	0.21680	0.11178
200	0.36834	0.26173	0.13551
250	0.41776	0.30127	0.15761
	Functional Off-site Application Rate[1]		
5	0.00e+00	0.00e+00	0.00e+00
10	3.12e-08	5.80e-03	7.50e-03
15	1.62e-02	1.79e-02	1.63e-02
20	3.16e-02	3.05e-02	2.37e-02
25	4.77e-02	4.25e-02	3.01e-02
50	1.22e-01	9.17e-02	5.34e-02
100	2.38e-01	1.65e-01	8.57e-02
150	3.09e-01	2.17e-01	1.12e-01
200	3.68e-01	2.62e-01	1.36e-01
250	4.18e-01	3.01e-01	1.58e-01
	Sensitive Species -Hazard Quotient[2]		
5	0.0e+00	0.0e+00	0.0e+00
10	9.4e-08	1.7e-02	2.3e-02
15	4.9e-02	5.4e-02	4.9e-02
20	9.5e-02	9.2e-02	7.1e-02
25	1.4e-01	1.3e-01	9.0e-02
50	3.7e-01	6.4e-01	1.6e-01
100	7.2e-01	4.9e-01	2.6e-01
150	9.3e-01	6.5e-01	3.4e-01
200	**1.1e+00**	7.9e-01	4.1e-01
250	**1.3e+00**	9.0e-01	4.7e-01

[1] The functional off-site application rate is calculated as the nominal application rate (specified above after the worksheet title) multiplied by the proportion lost in runoff.

[2] The hazard quotient is calculated as the functional off-site application rate divided by the NOEC value. The NOEC's are specified above on the lines following the application rate.

Table 4-10: Summary of exposure assessment and risk characterization for terrestrial plants from runoff for triclopyr bee.

Application rate	1	lb/acre	Typical FS rate, Section 2.4.
Sensitive Species (Lowest NOEC)	0.003	lb/acre	Section 4.3.2.4.

Annual Rainfall	Clay	Loam	Sand
	Proportion lost in Runoff		
5	0.00000	0.00000	0.00000
10	0.00000	0.00047	0.00235
15	0.00091	0.00183	0.00711
20	0.00202	0.00384	0.01307
25	0.00367	0.00619	0.01971
50	0.01329	0.02044	0.05591
100	0.03735	0.05449	0.13414
150	0.06379	0.09103	0.21388
200	0.09155	0.12877	0.25367
250	0.12016	0.16732	0.26940
	Functional Off-site Application Rate[1]		
5	0.00e+00	0.00e+00	0.00e+00
10	0.00e+00	4.74e-04	2.35e-03
15	9.10e-04	1.83e-03	7.11e-03
20	2.02e-03	3.84e-03	1.31e-02
25	3.67e-03	6.19e-03	1.97e-02
50	1.33e-02	2.04e-02	5.59e-02
100	3.73e-02	5.45e-02	1.34e-01
150	6.38e-02	9.10e-02	2.14e-01
200	9.15e-02	1.29e-01	2.54e-01
250	1.20e-01	1.67e-01	2.69e-01
	Sensitive Species -Hazard Quotient[2]		
5	0.0e+00	0.0e+00	0.0e+00
10	0.0e+00	1.6e-01	7.8e-01
15	3.0e-01	6.1e-01	**2**
20	6.7e-01	**1.3**	**4**
25	**1.2**	**2**	**7**
50	**4**	**7**	**19**
100	**12**	**18**	**45**
150	**21**	**30**	**71**
200	**31**	**43**	**85**
250	**40**	**56**	**90**

[1] The functional off-site application rate is calculated as the nominal application rate (specified above after the worksheet title) multiplied by the proportion lost in runoff.

[2] The hazard quotient is calculated as the functional off-site application rate divided by the NOEC value. The NOEC's are specified above on the lines following the application rate.

Table 4-11: Summary of exposure assessment and risk characterization for terrestrial plants from drift for triclopyr TEA.

	Most Sensitive Plant	
Post-emergence NOEC, lb/acre	0.0041	
Application Rate, lb/acre	1	See chemical specific notes
Estimates of the proportion of offsite drift		
Distance (feet)	Drift	Terrestrial Drift based on AGDRIFT using a low boom ground sprayer. See Worksheet A06 for details and drift estimates based on other methods of application.
25	0.0187	
50	0.0101	
100	0.0058	
300	0.0024	
500	0.0015	
900	0.0008	
Estimates of functional offsite application rate		
Distance (feet)	Rate (lb/acre)	
25	0.0187	Calculated as the product of the application rate and the estimated proportion of offsite drift.
50	0.0101	
100	0.0058	
300	0.0024	
500	0.0015	
900	0.0008	
Hazard Quotient - Sensitive Species		
25	5	Calculated as the offsite application rate divided by the NOEC for the most sensitive species.
50	2	
100	1.4	
300	5.9e-01	
500	3.7e-01	
900	2.0e-01	

Table 4-12: Summary of exposure assessment and risk characterization for terrestrial plants from drift for triclopyr BEE.

	Most Sensitive Plant	
Post-emergence NOEC, lb/acre	0.0039	
Application Rate, lb/acre	1	See chemical specific notes
Estimates of the proportion of offsite drift		
Distance (feet)	Drift	Terrestrial Drift based on AGDRIFT using a low boom ground sprayer. See Worksheet A06 for details and drift estimates based on other methods of application.
25	0.0187	
50	0.0101	
100	0.0058	
300	0.0024	
500	0.0015	
900	0.0008	
Estimates of functional offsite application rate		
Distance (feet)	Rate (lb/acre)	
25	0.0187	Calculated as the product of the application rate and the estimated proportion of offsite drift.
50	0.0101	
100	0.0058	
300	0.0024	
500	0.0015	
900	0.0008	
Hazard Quotient - Sensitive Species		
25	5	Calculated as the offsite application rate divided by the NOEC for the most sensitive species.
50	3	
100	1.5	
300	6.2e-01	
500	3.8e-01	
900	2.1e-01	

Table 4-12: Quantitative risk characterization for aquatic species from triclopyr TEA.

Hazard Quotients	**Central**	**Lower**	**Upper**	**Endpoint**
Fish				
Acute, Stream	5e-04	5e-06	2e-03	Mortality
Acute, Aquatic Weeds	8e-03	4e-03	1e-02	Mortality
Chronic	3e-04	8e-05	5e-04	NOEC
Aquatic Invertebrates				
Acute, Stream	6e-04	7e-06	3e-03	Mortality
Acute, Aquatic Weeds	1e-02	5e-03	2e-02	Mortality
Chronic	4e-04	1e-04	6e-04	NOEC
Aquatic Plants				
Acute, Stream	2e-02	2e-04	1e-01	Mortality
Acute, Aquatic Weeds	4e-01	2e-01	6e-01	
Chronic	7e-03	2e-03	1e-02	

Exposures (mg/L)	Central	Lower	Upper	Worksheet
Acute, Stream	0.09	0.001	0.4	F06
Acute, Aquatic Weeds	1.5	0.75	2.5	Section 3.2.3.4.4
Longer-term [1]	0.03	0.008	0.05	F09

Toxicity values (mg/L)			
	Value (mg/L)	Endpoint	Section
Fish, acute	199	LC_{50}	4.3.3.
Fish, chronic	104	NOEC	
Aquatic Invertebrates, acute	139	LC_{50}	
Aquatic Invertebrates, chronic	80.7	NOEC	
Aquatic plants	4.2	LC_{50}	

Table 4-13: Quantitative risk characterization for aquatic species from triclopyr BEE.

Hazard Quotients	**Central**	**Lower**	**Upper**	**Endpoint**
Fish				
Acute	6e-02	1e-03	6e-01	Mortality
Chronic	See Table 4-13 for triclopyr TEA			
Aquatic Invertebrates				
Acute	2e-03	4e-05	2e-02	Mortality
Chronic	See Table 4-13 for triclopyr TEA			
Aquatic Plants				
Acute	2e-01	4e-03	**2**	EC_{50}
Chronic	See Table 4-13 for triclopyr TEA			

Exposures (mg/L)	Central	Lower	Upper	Worksheet	
Acute	0.014	0.0003	0.15	F06	Stream [1]
Longer-term	See Table 4-13 for triclopyr TEA				

Toxicity values (mg/L)			
	Value (mg/L)	Endpoint	Section
Fish, acute	0.25	LC_{50}	4.3.3.
Fish, chronic	See Table 4-13 for triclopyr TEA		
Aquatic Invertebrates, acute	8.55	LC_{50}	
Aquatic Invertebrates, chronic	See Table 4-13 for triclopyr TEA		
Aquatic plants	0.07	NOEC	

[1] Based on GLEAMS modeling of maximum concentrations of triclopyr.

5. LIST OF WORKS CONSULTS

ATSDR (Agency for Toxic Substances and Disease Registry). 2002. ATSDR ToxProfiles on CD-ROM. Available from U.S. Department of Health and Human Services, Public Health Service, ATSDR, Division of Toxicology. http://www.atsdr.cdc.gov/.

Abdelghani AA; Tchounwou PB; Anderson AC; Sujono H; Heyer LR; Monkiedje A. 1997. Toxicity evaluation of single and chemical mixtures of Roundup, Garlon-3a, 2,4-D, and syndets surfactant to channel catfish (Ictalurus punctatus), bluegill sunfish (Lepomis microchirus), and crawfish (Procambarus spp.). Environmental Toxicology and Water Quality; 12 (3): 237-243.

Abdelghani AA. 1995. Assessment of the exposure of workers applying herbicide mixtures (2, 4-D+Roundup, Garlon-3a+Roundup), toxicity and fate of these mixtures in the environment. summary report. NTIS/PB96-179221.

Alden CL; Frith CH. 1991. The Urinary System. Chapter 15 in :Handbook of Toxicologic Pathology. WM Haschek and CG Rousseaux, editors. Academic Press, New York, NY. pp. 316-381.

Alexander H. 1993. Supplemental to MRID No. 41969902--Garlon 4 Herbicide: Acute Flow-through Toxicity to Grass Shrimp, Palaemonetes pugio: Lab Project Number: MWM093093. Unpublished study prepared by Dow Chemical Co., Environmental Tox. & Chem. Research Lab and DowElanco. .

Allen RR; Fryrear DW. 1977. Limited tillage saves soil, water, and energy. ASAE Annual Meeting, NC State Univ., Raleigh, NC. June 26-29, 1977. 14 pp.

Anthony DC; Montine TJ; Graham DG. 1996. Toxic Responses of the Nervous System. In: Casarett and Doull's Toxicology: The Basic Science of Poisons. 5th Edition. McGraw-Hill, Health Professions Division, New York, NY. pp. 463-486.

Barna-Lloyd T; Yano B; Rachunek B. 1992. Triclypyr Butoxyethyl Ester (Tricloypyr Bee): Subchronic Toxicity Study in Fischer 344 Rats: Lab Project Number: K-120085-011. Unpublished study prepared by The Dow Chemical Co. 215 p. (Tricloypyr Bee): Subchronic Toxicity Study in Fischer 344 Rats: Lab Project Number: K-120085-011. Unpublished study prepared by The Dow Chemical Co. 215 p.

Barron M; Ball T. 1989. Garlon 3A Herbicide: Evaluation of the Toxicity to the Chennel Catfish(Ictalurus punctatus):. Lab Project Number: ES-DR-0121-6064-8: ES-2097. Unpublished study pre- pared by The Dow Chemical Co. 15 p.

Barron M; Mayes M; Ball T. 1989. Garlon 3A Herbicide Evaluation of The Toxicity to The Crayfish: Lab Project Number: ES-DR- 0121-6064-78. Unpublished study prepared by The Dow Chemical Co. 15 p. .

Barron MG; Mayes MA; Murphy PG; Nolan RJ. 1990. Pharmacokinetics and metabolism of triclopyr butoxyethyl ester in Coho salmon. Aquat Toxicol (Amst); 16 (1): 19-32.

Barron MG; Hansen SC; Ball T. 1991. Pharmacokinetics and metabolism of triclopyr in the crayfish (Procambarus clarki). Drug Metab Dispos. 19(1): 163-7.

Batchelder T; McCarty W. 1977. Static Acute Toxicity of Triclopyr to Daphnia magna, Letter Report ES-37L, Environmental Sciences Research. Unpublished study prepared by Dow Chemical Co. 3 p. .

Beavers JB; Fink R; Grimes J; et al. 1979. Final Report: One-Generation Reproduction Study--Bobwhite Quail: Project No. 103-191. (Unpublished study received Apr 29, 1980 under 434-546; prepared by Wildlife International, Ltd., submitted by Dow Chemical U.S.A., Midland, Mich.; CDL:242368-D).

Beavers JB; Fink R; Grimes J; et al. 1980. Final Report: One-Generation Reproduction Study--Mallard Duck: Project No. 103-192. (Unpublished study received Apr 29, 1980 under 464-546; prepared by Wildlife International, Ltd., submitted by Dow Chemical U.S.A., Midland, Mich.; CDL:242368-C).

Becker J. 2000. Validation of a Method for the Analysis of Triclopyr Butoxyethyl Ester and Impurities in Triclopyr Butoxyethyl Ester Technical Grade of Active Ingredient: Lab Project Number: DECO GL-AL MD-2000-00476. Unpublished study prepared by The Dow Chemical Compa. .

Beliles RP; Wosu NJ. 1976. Three Generation Reproduction Study in Rats: Dowco 233: LBI Project No. 2528. Final rept. (Unpublished study received May 10, 1977 under 464-546; prepared by Litton Bionetics, Inc., submitted by Dow Chemical U.S.A., Midland, Mich.; CDL:229780-F).

Bentson KP; Norris LA. 1991. Foliar penetration and dissipation of triclopyr butoxyethyl ester herbicide on leaves and glass slides in the light and dark. J Agric Food Chem. 39 (3) :622-630.

Berdasco N. 1990. 15(percent) Dilution of Garlon 3A: Dermal Sensitization Potential in the Hartley Albino Guinea Pig: Lab Project No. M-003724-013: M-004714-008. Unpublished study pre- pared by Dow Chemical Co. 14 p. 5(percent) Dilution of Garlon 3A: Dermal Sensitization Potential in the Hartley Albino Guinea Pig: Lab Project No. M-003724-013: M-004714-008. Unpublished study pre- pared by Dow Chemical Co. 14 p.

Berdasco N. 1990. 30(percent) Dilution of Garlon 3A: Dermal Sensitization Potential in the Hartley Albino Guinea Pig: Lab Project: M-003724-012: M-004714-008. Unpublished study prepared by Chemical Co. 14 p. 0(percent) Dilution of Garlon 3A: Dermal Sensitization Potential in the Hartley Albino Guinea Pig: Lab Project: M-003724-012: M-004714-008. Unpublished study prepared by Chemical Co. 14 p.

Berdasco N. 1990. 2.5(percent) Dilution of Garlon 4: Dermal Sensitization Potential in the Hartley Albino Guinea Pig: Lab Project No. M-004714-008. Unpublished study prepared by Dow Chemical Co. 14 p. 5(percent) Dilution of Garlon 4: Dermal Sensitization Potential in the Hartley Albino Guinea Pig: Lab Project No. M-004714-008. Unpublished study prepared by Dow Chemical Co. 14 p.

Berdasco N. 1990. 7.5(percent) Dilution of Garlon 4: Dermal Sensitization Potential in the Hartley Albino Guinea Pig: Lab Project No. M-004714-007: M-004714-008. Unpublished study pre- pared by Chemical Co. 14 p. 5(percent) Dilution of Garlon 4: Dermal Sensitization Potential in the Hartley Albino Guinea Pig: Lab Project No. M-004714-007: M-004714-008. Unpublished study pre- pared by Chemical Co. 14 p.

Berdasco N. 1994. Four Samples of Garlon 3A: Dermal Sensitization Potential in the Hartley Albino Guinea Pig: Lab Project Number: M/003724/011E1: M/004714/006E1. Unpublished study prepared by The Toxicology Research Lab. 21 p. .

Berdasco N. 1994. Four Samples of Garlon 4: Dermal Sensitization Potential in the Hartley Albino Guinea Pig: Lab Project Number: M/004714/006E1. Unpublished study prepared by The Toxicology Research Lab. 21 p. .

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

Blevins C. 2001. Product Chemistry Analysis of Triclopyr Product Identity, Composition and Analysis: Amended Final Report: Lab Project Number: 310045.1.039. Unpublished study prepared by Midwest Research Institute. 101 p. {OPPTS 830.1700 and 830.1800}. .

Blevins C. 2002. Group B: Physical and Chemical Properties Testing of Triclopyr Triethylamine Salt: Lab Project Number: 310045.1.037.02. Unpublished study prepared by Midwest Research Institute. 16 p. {OPPTS 830.6317, 830.6320}. .

Boggs JF; Mcmurry ST; Leslie Dm Jr; Engle DM; Lochmiller RL. 1990. Influence of habitat modification on the intestinal helminth community ecology of cottontail rabbit populations. J Wildl Dis. 26(2):157-69.

Boggs JF; Lochmiller RL; Mcmurry ST; Leslie D M JR; Engle DM. 1991. Cuterebra infestations in small-mammal communities as influenced by herbicides and fire. J Mammal. 72 (2): 322-327.

Boggs JF; Mcmurry ST; Leslie Dm Jr; Engle DM; Lochmiller RL. 1991b. Influence of habitat modification on the community of gastrointestinal helminths of cotton rats. J Wildl Dis. 27(4): 584-93.

Bollig JJ; Seiler JR; Zedaker SM; Thompson JW; Lucero D. 1995. Effect of plant moisture stress and application surface on uptake and translocation to triclopyr with organosilicone surfactant in red maple seedlings. Canadian Journal of Forest Research; 25 (3): 425-429.

Boren JC; Lochmiller RL; Leslie DM; Engle DM. 1993. Long-term effects of woody vegetation management on seasonal body condition of northern bobwhites. J Range Manag. 46: 520-523.

Borrecco J; Neisess J. 1991. Risk assessment for the impurities2-butoxyethanol and 1,4-dioxane found in Garlon 4 and Roundup herbicide formulations. Pacific Southwest Region, Forest Pest Management. ReportNo. R91-2. 33 pages.

Boulding JR. 1995. Practical Handbook of Soil, Vadose Zone, and Ground-Water Contamination. Lewis Publishers, Boca Raton, Florida, 948 pp.

Bovey RW; Meyer RE. 1981. Effect of 2,4,5-t, triclopyr and 3,6-dichloropicolinic acid on crop seedlings. Weed Sci. 29 (3): 256-261.

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

Boyd RS; Miller JH. 1997. Forest herbicide site preparation treatments have little impact on plant diversity 11 years posttreatment. Bull Ecol Soc Amer. 78 (Suppl. 4): 58.

Branham BE; Lickfeldt DW. 1997. Effect of pesticide-treated grass clippings used as a mulch on ornamental plants. Hortscience. 32 (7): 1216-1219.

Breslin WJ; Billington R. 1995. Evaluation of the developmental toxicity of triclopyr triethylamine salt (Tea) and triclopyr butoxyethyl ester (Bee) in rabbits. International Toxicologist. 7(1):ab# 74-p-5.

Breslin WJ; Billington R; Jones K. 1996. Evaluation of the developmental toxicity of triclopyr triethylamine salt (TTEA) and triclopyr butoxyethyl ester (TBEE) in rats. Teratology. 53(2):106.

Breslin W. 1990a. Results of a Teratology Study on DOWCO 233in the Rat:. Lab Project Number: K-042085-05. Unpublished study prepared by The Dow Chemical Co. 165 p.

Breslin W. 1990b. Dow Chemical U S A Phase 3 Summary of MRID 00072441. Teratogenicity - Rat: Results of a Teratology Study on Dowco 233 [. [(3,5,6-Trichloro-2-Pyridyloxy) Acetic Acid] in the Rat: Lab ID: K-042085-51; NBX-238. Prepared by The Dow Chemical Company. 10 p.

Breslin W. 1990c. Dow Chemical U S A Phase 3 Summary of MRID 41200303. Teratogenicity - Rabbit Dowco 233: Oral Teratology Study in New Zealand White Rabbits: Lab ID. K-042085-042. Prepared by Dow Chemical U S A. 12 p. .

Bromilow, RH; Chamberlain, K; Evans, AA. 1990. Physicochemical aspects of phloem translocation of herbicides. Weed Sci. 38: 305-314.

Brooks K; DeWildt P. 2000. NAF-5 (Pathfinder II Herbicide): Acute Dermal Toxicity Study in Fischer 344 Rats: Lab Project Number: 001078. Unpublished study prepared by The Dow Chemical Co. 45 p. {OPPTS 870.1200}. (Pathfinder II Herbicide): Acute Dermal Toxicity Study in Fischer 344 Rats: Lab Project Number: 001078. Unpublished study prepared by The Dow Chemical Co. 45 p. {OPPTS 870.1200}.

Brown CD; Hollis JM. 1996. Swat: a semi-empirical model to predict concentrations of pesticides entering surface waters from agricultural land. Pesticide Science; 47 (1) 1996 41-50.

Brown A. 2001. Triclopyr 13.6% Ester; Physical and Chemical Characteristics: Lab Project Number: MI-0107. Unpublished study prepared by Micro Flo Company. 8 p. {OPPTS 830.6303, 830.7300, 830.7000, 830.6315, 830.7100, 830.6319, 830.6320}. .

Brudenell A JP; Baker DA; Grayson BT. 1995. Phloem mobility of xenobiotics: tabular review of pH properties governing the output of the kleier model. Plant Growth Regulation. 16 (3): 215-231.

Bryson A. 1994a. Triclopyr Butoxyethyl Ester: A Study of the Effect on Pregnancy of the Rabbit: Lab Project Number: DWC 650/643/931352. Unpublished study prepared by Huntingdon Research Centre Ltd. 153 p. .

Bryson A. 1994b. Triclopyr Triethylamine Salt: A Study of the Effect on Pregnancy of the Rat: Lab Project Number: DWC 645/646/931358. Unpublished study prepared by Huntingdon Research Centre Ltd. 171 p. .

Bryson A. 1994c. Triclopyr Triethylamine Salt: A Study of the Effect on Pregnancy of the Rabbit: Lab Project Number: 642/641/659/931914. Unpublished study prepared by Huntingdon Research Centre Ltd. 181 p. .

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

Buick RD; Robson B; Field RJ. 1992. A mechanistic model to describe organosilicon surfactant promotion of triclopyr uptake. Pestic Sci. 36 (2): 127-133.

Burnmaster DE. 1998. Lognormal distribution for total water intake and tap water intake by pregnant and lactating women in the United States. Risk Analysis. 18(5): 215-219.

Burns LA; Meade BJ; Munson AE. 1996. Toxic Responses of the Immune System. In: Casarett and Doull's Toxicology: The Basic Science of Poisons. 5th Edition. McGraw-Hill, Health Professions Division, New York, NY. pp. 355-402.

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

Bush PB; Neary DG; Mcmahon CK; Taylor J W JR. 1987. Suitability of hardwoods treated with pH and pyridine herbicides for use as firewood. Arch Environ Contam Toxicol. 16 (3): 333-342.

C&P Press (Chemical and Pharmaceutical Press). 2002. Greenbook.net. http://www.greenbook.net/.

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

Calabrese EJ. 1991. Principles of animal extrapolation. Lewis Publishers. Chelsea, MI.

Campbell S; Lynn S. 1991. Garlon 4 Herbicide: An Acute Oral Toxicity Study With the Northern Bobwhite: Lab Project Number: ES- DR-0224-6186-8. Unpublished study prepared by Wildlife Interna- tional LTD. 22 p.

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

Carmichael N; Perkins J; Nolan R et al. 1988. Human Dermal Absorption Study of Garlon-4 (A Herbicide): Laboratory Project ID 87/DCS041/835. Unpublished study performed by Dow Chemical Co. in association with CTC. (International) Ltd. and Charter- house Clinical Research Unit Ltd. 95 p.

Carmichael NG; Nolan RJ; Perkins JM; Davies R; Warrington SJ. 1989. Oral and dermal phamacokinetics of triclopyr in human volunteers. Hum Toxicol. 8(6):431-7.

Carmichael NG. 1989. Assessment of hazards to workers applying pesticides. Food Addit Contam. Suppl 1: S21-7.

Carreon R. 1985. Garlon 3A: Dermal Sensitization Potential in the Guinea Pig: Laboratory Project ID; HET M-003724-008. Unpublished study prepared by Dow Chemical U.S.A. 11 p.

Carter L. 2000. Acute Inhalation Toxicity Study in Rats: Triclopyr: Final Report: Lab Project Number: 5831-00. Unpublished study prepared by Stillmeadow, Inc. 19 p. {OPPTS 870.1300}.

Chakravarty P; Sidhu SS. 1987. Effect of glyphosate hexazinone and triclopyr on in-vitro growth of five species of ectomycorrhizal fungi. Eur J for Pathol. 17 (4-5): 204-210.

Chow C. 1988. Analysis of Garlon 3A Herbicide for N-nitrosodi-ethylamine: Laboratory Product ID: ML-AL 87-50037. Unpublished study prepared by Dow Chemical Co. 5 p.

Coffman CB; Frank JR; Potts WE. 1993. Crop responses to Hexazinone, Imazapyr, Tebuthiuron, and Triclopyr. Weed Tech. 7: 140-145.

Concha M; Kennard L. 1997. Photodegradation of (2,6-(carbon 14))Triclopyr in/on Soil by Natural Sunlight:. (Final Report): Lab Project Number: 647W-1: 647W: ENV 97064. Unpublished study prepared by PTRL West, Inc. 110 p.

Coupe RH; Thurman EM; Zimmerman LR. 1998. Relation of usage to the occurrence of cotton and rice herbicides in three streams of the Mississippi Delta. Environmental Science & Technology; 32 (23) 1998 3673-3680.

Cowgill U; Milazzo D; Landenberger B. 1988. A Comparison of the Effect of Triclopyr Triethylamine Salt(Garlon 3A) on Two Species of Duckweed Examined For Seven-Day and Fourteen Day Period:. Lab Project Number: ES-DR-0003-7070-2. Unpublished study prepared by Dow Chemical Co. 49 p.

Cowgill UM; Milazzo DP; Landenberger BD. 1989a. A comparison of the effect of triclopyr triethylamine salt on two species of duckweed lemna examined for a 7 and 14-day test period. Water Res; 23 (5): 617-624.

Cowgill UM; Milazzo DP; Landenberger BD. 1989b. Toxicity of nine benchmark chemicals to Skeletonema costatum, a marine diatom. Environ Toxicol Chem. 8 (5): 451-45.

Cox C. 2000. Triclopyr Herbicide Factsheet. J Pesticide Reform. 20(4): 12-19.

Derr JF. 1993. Tolerance of apple and peach trees to triclopyr. Hortscience. 28 (10): 1021-1023.

Deubert KH; Corte-Real I. 1986. Soil residues of picloram and triclopyr after selective foliar application on utility rights-of-way. J Arboric. 12 (11): 269-272.

Diaz-Diaz R; Loague K. 2001. Assessing the potential for pesticide leaching for the pine forest areas of Tenerife. Environ Toxicol Chem. 20(9):1958-67.

Dow AgroSciences LLC. 1999. Submission of Product Chemistry Data in Support of the Reregistration of the Triclopyr Containing Products Garlon 3A and Redeem, Hammer and Triclopyr Triethylamine Salt Solution, Turflon II Amine, Confront, Grandstand R, XRM-5202, Dow AgroSicences Brush.

Dow AgroSciences LLC. 2002. Revised Product Label for Garlon 3A. Dow AgroSciences LLC, Indianopolis, IN 46268. Label Code: D02-101-035, Accepted by EPA on 12/03/2002.

Dowdy DL; Mckone TE; Hsieh D PH. 1996. Prediction of chemical biotransfer of organic chemicals from cattle diet into beef and milk using the molecular connectivity index. Environmental Science and Technology. 30 (3): 984-989.

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

Eckerlin RH; Ebel Jg Jr; Maylin GA; Muscato TV; Gutenmann WH; Bache CA; Lisk DJ. 1987. Excretion of triclopyr herbicide in the bovine. Bull Environ Contam Toxicol. 39(3):443-7.

Eisenbrandt D. 1988. Response to: Memorandum (April 28, 1988) and Data Evaluation Reports, Toxicology Branch, Environmental Pro- tection Agency--Subject: Triclopyr Herbicide--Review of Chronic Toxicity and Carcinogenicity Studies in Rats (MRID No. 40107701) and Mice. (MRID No. 40356601): Laboratory ID: K-040285-026. Un- published study prepared by Dow Chemical Co. 86 p.

Eisenbrandt D. 1990. Dow Chemical U S A Phase 3 Summary of MRID 40200302. Response to: Memorandum. (April 28, 1988) and Data Evaluation Reports, Toxicology Branch, Environmental Protection Agency-- Subject: Triclopyr Herbicide - Review of Chronic Toxicity and Carcinogenicity Studies in Rats.. and Mice..: Project ID: K-040285-026. Prepared by DOW CH.

Eisenbrandt D; Firchau H; Wolfe E; et al. 1987. Triclopyr: 2-year Dietary Chronic Toxicity-oncogenicity Study in Fischer 344 Rats: Final Report: Laboratory Project No. HET K-042085-026. Unpublished study prepared by Dow Chemical Co. 1094 p.

Eisenbrandt D; Nolan R; McMaster S et al. 1997. Triclopyr: An Assessment of Common Mechanism of Toxicity: Lab Project Number: HET K-042085-097. Unpublished study prepared by The Dow Chemical Co. 15 p.

Engle DM; Stritzke JF; McCollum FE. 1991. Vegetation management in the Cross Timbers: response of understory vegetation to herbicides and burning. Weed Tech. 5: 406-410.

Estok D; Freedman B; Boyle D. 1989. Effects of the herbicides 2 4-d glyphosate hexazinone and triclopyr on the growth of three species of ectomycorrhizal fungi. Bull Environ Contam Toxicol. 42 (6): 835-839.

Feldmann RJ; Maibach HI. 1974. Percutaneous penetration of some pesticides and herbicides in man. Toxicol. Appl. Pharmacol. 28: 126-132.

Feng Y; Minard RD; Bollag J-M. 1998. Photolytic and microbial degradation of 3,5,6-trichloro-2-pyridinol. Environmental Toxicology and Chemistry; 17 (5): 814-819.

Finco D; Cooper T. 1995. Effects of Triclopyr on Phenolsulphonphthalein. (PSP) Excretion and Creatinine Clearance in Dogs: Lab Project Number: K-042085-059. Unpublished study prepared by The University of Georgia, College of Veterinary Medicine. 20 p.

Finney DJ. 1971. Probit Analysis. New York: Cambridge University Press. 333 p.

Fischer JB; Michael JL. 1997. Use of elisa immunoassay kits as a complement to hplc analysis of imazapyr and triclopyr in water samples from forest watersheds. Bulletin of Environmental Contamination and Toxicology. 59 (4): 611-618.

Fletcher JS; Nellessen JE; Pfleeger TG. 1994. Literature review and evaluation of the EPA food-chain (Kenega) nomogram, an instrument for estimating pesticide residues on plants. Environ. Toxicol. Chem. 13(9):1383-1391.

Fontaine D. 1990. Dispersal and Degradation of Triclopyr within a Canadian Boreal Forest Ecosystem following an Aerial Application of Garlon 4: Lab Project Number: GH-C 2314: 87066. Unpublished study prepared by DowElanco, North American Environmental Chemi- stry Lab and.

Foster D; Getsinger K; Petty D. 1997. The Aquatic Dissipation of Triclopyr in a Whole-Pond Treatment: Lab Project Number: ENV95012. Unpublished study prepared by DowElanco, ABC Labs. and A&L Great Lakes Lab. 306 p. {OPPTS 860.1400}.

Gabor ST; Haagsma T; Murkin HR; Armson E. 1995. Effects of triclopyr amine on purple loosestrife and non-target wetland plants in south-eastern ontario, canada. Journal of Aquatic Plant Management. 33: 48-51.

Gambini GF; Mantovani C; Pira E; Piolatto PG; Negri E. 1997. Cancer mortality among rice growers in Novara Province, northern Italy. American Journal of Industrial Medicine. 31 (4): 435-441.

Gardner R. 1996. Response to EPA Review of Triclopyr Terrestrial and Forestry Field Dissipation Studies: "Non-Crop Right-of-way Terrestrial Dissipation of Triclopyr in California:" MRID 42730601: and "The Dissipation and Movement of Triclopyr in a Northern USA Forest Sys.

Gardner SC; Grue CE. 1996. Effects of Rodeo and Garlon 3a on nontarget wetland species in central Washington. Environmental Toxicology and Chemistry. 15 (4): 441-451.

Gardner SC; Grue CE; Grassley JM; Lenz LA; Lindenauer JM; Seeley ME. 1997. Single species algal Ankistrodesmus toxicity tests with Rodeo and Garlon 3a. Bulletin of Environmental Contamination and Toxicology. 59 (3): 492-499.

Garry VF; Burroughs B; Tarone R; Kesner JS. 1999. Herbicides and adjuvants: an evolving view. Toxicol Ind Health. 15(1-2):159-67.

Gaskins M. 2001. Triclopyr 13.6% Ester Product Chemistry: Lab Project Number: MI-0107. Unpublished study prepared by Micro Flo Co. 34 p. {OPPTS 830.1550, 830.1600, 830.1650, 830.1670, 830.1700, 830.1750}.

Gehring PJ; Kramer CG; Schwetz BA; Rose JQ; Rowe VK. 1973. The fate of 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) following oral administration to man. Toxicol Appl Pharmacol. 26: 352-361.

Gersich F; Mandoza C; Hopkins D; et al. 1982. The Acute and Chronic Toxicity of Triclopyr Triethylamine Salt Solution to Daphnia magna Straus: ES-583. Unpublished study prepared by Environmental Sciences Research, Dow Chemical U.S.A. 19 p. (..) Triethylamine Salt Solution to Daphnia magna Straus: ES-583. Unpublished study prepared by Environmental Sciences Research, Dow Chemical U.S.A. 19 p.

Gersich FM; Mendoza CG; Hopkins DL; Bodner KM. 1984. Acute and chronic toxicity of triclopyr triethylamine salt to daphnia magna straus. Bull Environ Contam Toxicol. 32(4):497-502.

Gersich F; Hopkins D; Milazzo D. 1985. The Development of Flow-through Acute and Chronic Test Methods for Daphnia magna Straus: ES-756. Unpublished study prepared by Dow Chemical U.S.A. 20 p.

Getsinger KD; Madsen JD; Netherland MD; Turner EG. 1996. Field evaluation of triclopyr (Garlon 3a) for controlling eurasian watermilfoil in the pend oreille river, washington. NTIS/AD-A304 807/1.

Getsinger KD; Turner EG; Madsen JD; Netherland MD. 1997. Restoring native vegetation in a eurasian water milfoil-dominated plant community using the herbicide triclopyr. Regulated Rivers Research and Management; 13 (4). 1997. 357-375.

Getsinger, K. D., et al. 2000. Aquatic dissipation of the herbicide triclopyr in Lake Minnetonka, Minnesota. Pest Manag Sci. 56:388-400.

Ghaoui L. 1999. Group B-Physical/Chemical Properties for Garlon 4, an Emulsifiable Concentrate Formulation Containing 62.1% Triclopyr-2-butoxyethyl Ester: Lab Project Number: NAFST057. Unpublished study prepared by Dow AgroSciences LLC. 5 p. {OPPTS 830.6302, 830.6303,.

Gilbert K. 1996. Garlon 3A: Acute Dermal Toxicity Study in New Zealand White Rabbits: Lab Project Number: M-003724-015D. Unpublished study prepared by Dow Chemical Co. Health and Environmental Sciences. 31 p.

Goetz CG; Bolla KI; Rogers SM. 1994. Neurologic health outcomes and Agent Orange: Institute of Medicine report. Neurology. 44(5): 801-809.

Goldstein RS; Schnellmann RG. 1996. Toxic Responses of the Kidney. In: Cassarett and Doull's Toxicology: The Basic Science of Poisons. 5th ed., CD Klaassen, MO Amdur, J Doull, Eds. McGraw-Hill, New York, NY. pp. 417-442.

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

Goodman D; Hildebrandt P. 1988. Review of Tumor Data From the Two-Year Dietary Chronic Toxicity/Oncogenicity Study on Triclopyr in F344 Rats: Lab Project Number: MLJ61495. Unpublished study prepared by PATHCO, Inc. 94 p.

Goodman D; Hildebrandt P. 1996. Review of Tumor Data from the Chronic Toxicity/Carcinogenicity Studies of Triclopyr in F344 Rats and ICR and ICR(Jcl:ICR) Mice. Lab Project Number: 96-86. Unpublished study prepared by Pathco, Inc. 489 p.

Gorzinski S; Lehr K; Piasecki D; et al. 1991. Garlon 4 Herbicide: Static Acute 96-Hour Toxicity to the Rainbow Trout, Oncorhynchus mykiss Walbaum: Lab Project Number: ES-DR-022406168-4. Unpublished study prepared by DowElanco. 33 p.

Greaves S. 2001. Triclopyr Butoxyethyl Ester Technical Product Identity and Composition, Description of Materials Used to Produce the Product, Description of Production Process and Discussion of Impurities: Lab Project Number: 310045.1.036.01. Unpublished study prepared.

Groninger JW; Stein HD; Zedaker SM; Smith DW. 1998. Growth response and cost comparisons for precommercial thinning methods of appalachian oak stump sprout clumps. Southern Journal of Applied Forestry; 22 (1) 1998 19-23.

Hall S. 1999. Group B-Physical/Chemical Properties for Garlon 3A, A Liquid End-Use Product Containing Triclopyr TEA: Lab Project Number: NAFST066. Unpublished study prepared by Dow AgroSciences LLC. 5 p. {OPPTS 830.6302, 830.6303, 830.6304, 830.6314, 830.6315, 830.6.

Hall S; Chamberlain J; Godwin-Saad E. 1995. Effects of pollutants on freshwater organisms. Water Environment Research. 67 (4): 713-718.

Hanley T; Thompson D; Palmer A; et al. 1983. Teratology and Reproduction Studies with Triclopyr in the Rat and Rabbit. (Un- published study received Feb 17, 1984 under 464-546; submitted by Dow Chemical U.S.A., Midland, MI; CDL:072371-A).

Hanley Tr JR; Thompson DJ; Palmer AK; Beliles RP; Schwetz BA. 1984. Teratology and reproduction studies with triclopyr in the rat and rabbit. Fundam Appl Toxicol. 4:872-882,1984.

Harpole DN; Haas CA. 1999. Effects of seven silvicultural treatments on terrestrial salamanders. Forest Ecology and Management. 114:349-356.

Harrington TB; Wagner RG; Radosevich SR; Walstad JD. 1995. Interspecific competition and herbicide injury influence 10-year responses of CoA douglas-fir associated vegetation to release treatments. Forest Ecology and Management. 76 (1-3): 55-67.

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

Haschek WM; Rousseaux CG (Editors). 1991. Handbook of Toxicologic Pathology. Academic Press, New York, NY.

Hatterman-Valenti H; Christians NE; Owen M DK. 1995. Effect of 2,4-D and triclopyr on annual bedding plants. Journal of Environmental Horticulture. 13 (3): 122-125.

Havens P. 1995. Response to EPA Review EFGWB#92-0111: "Dispersal and Degradation of Triclopyr within a Canadian Boreal Forest Ecosystem Following an Aerial Application of Garlon 4:" MRID 41445001: Lab Project Number: GH-C 2314A. Unpublished study prepared by DowElanco E.

Hill R. 1999a. Product Identity, Composition, and Analysis for Triclopyr Triethylamine Salt Solution. (XRM-3724); an End-Use Product Containing Triclopyr Triethylamine Salt: Lab Project Number: NAFST080: DECO GL-AL 98-003941. Unpublished study prepared by Dow AgroSciences LLC. 122 p. {OPPTS 830.1550, 830.1600, 830.1650, 830.1800}.

Hill R. 1999b. Group A-Product Identity, Composition, and Analysis for Garlon 4 Herbicide (XRM-4714);. An End-Use Product Containing Triclopyr 2-Butoxyethyl Ester: Lab Project Number: NAFSTO73: DECO GL-AL 98-002794. Unpublished study prepared by Dow AgroSciences LLC. 95 p. {OPPTS 830.1550, 830.1600, 830.1670, 830.1700, 830.1750, 830.1800}.

Hill R. 2000. Triclopyr Triethyl Amine: Supplemental Production Assay Information: Lab Project Number: NAFST 312. Unpublished study prepared by Dow AgroSciences LLC. 15 p. {OPPTS 830.1700}.

Hill R. 2001. Group A-Product Identity, Composition, and Analysis for Garlon*4 Herbicide. (XRM4714); an End-Use Product Containing Triclopyr 2-Butoxyethyl Ester: Lab Project Number: NAFST073: DECO GL-AL 98-002794. Unpublished study prepared by Dow AgroSciences LLC. 95 p. {OPPTS 830.1550, 830.1600, 830.1650, 830.1670, 830.1750, 830.1800}.

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

Holmes SB; Thompson DG; Wainio-Keizer KL; Capell SS; Staznik B. 1994. Effects of lethal and sublethal concentrations of the herbicide, triclopyr butoxyethyl ester, in the diet of zebra finches. J Wildl Dis. 30(3):319-27.

Hotchkiss SA; Hewitt P; Caldwell J; Chen WL; Rowe RR. 1992. Percutaneous absorption of nicotinic acid, pH benzoic acid and triclopyr butoxyethyl ester through rat and human skin in vitro: further validation of an in vitro model by comparison with in vivo data. Food Chem Toxicol. 30(10):891-9.

Houtman B; Mayes M. 1997. Avian and Mammalian Risk Assessment for Triclopyr: Lab Project Number: GH-C 4354. Unpublished study prepared by DowElanco. 80 p.

Houtman B; Foster D; Getsinger K et al. 1997a. Aquatic Dissipation of Triclopyr in Lake Minnetonka, Minnesota: Lab Project Number: ENV94001: CMXX-94-0380: 13939. Unpublished study prepared by DowElanco, Braun Intertec and The Dow Chemical Co. 527 p. {OPPTS 860.1400}.

Houtman B; Foster D; Getsinger K et al. 1997b. Triclopyr Dissipation and the Formation and Decline of its TMP and TCP Metabolites in an Aquatic Environment: Lab Project Number: ENV96052: DE-05-96. Unpublished study prepared by DowElanco, A&L Great Lakes Labs., Inc. and Enviro-Bio-Tech, Ltd. 259 p.

Houtman B; Getsinger K; Petty D. 1997c. A Review of the Aquatic Environmental Fate of Triclopyr and its Major Metabolites: Lab Project Number: GH-C 4536. Unpublished study prepared by DowElanco. 29 p.

Houtman B. 1996. A Review of the Aquatic Dissipation Potential of Triclopyr in Transient, Non-Flowing Waterbodies Associated with the Use of Garlon 3A: Lab Project Number: GH-C 4275. Unpublished study prepared by DowElanco. 13 p.

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

Ioannou Y. 1990. Dow Chemical U S A Phase 3 Summary of MRID 40107701 and Related MRIDs 40356601. Triclopyr Herbicide -Review of Chronic Toxicity and Carcinogenicity Studies in Rats and Mice: DowElanco Project ID: K-040285-026; HET-K-042085-026. Prepared by DOW CHEMICAL.

Janz DM; Farrell AP; Morgan JD; Vigers GA. 1991. Acute pH stress responses of juvenile Coho salmon (Oncorhynchus kisutch) to sublethal concentrations of Garlon 4, Garlon 3a and Vision herbicides. Environ Toxicol Chem. 10 (1): 81-90.

Johansen JA; Geen GH. 1990. Sublethal and acute toxicity of the ethylene glycol butyl ether ester formulation of triclopyr to juvenile Coho salmon (Oncorhynchus kisutch). Arch Environ Contam Toxicol. 19(4): 610-616.

Johnson BD; Hall JC. 1996. Fluroxypryr- and triclopyr-specific enzyme-linked immunosorbent assays: Development and quantitation in soil and water. J. Agric. Food Chem. 44(2): 488-496.

Johnson WG; Lavy TL. 1994. In-situ dissipation of benomyl, carbofuran, thiobencarb, and triclopyr at three soil depths. Journal of Environmental Quality. 23 (3): 556-562.

Johnson WG; Lavy TL; Gbur EE. 1995a. Sorption, mobility and degradation of triclopyr and 2,4-D on four soils. Weed Science. 43 (4): 678-684.

Johnson WG; Lavy TL; Gbur EE. 1995b. Persistence of triclopyr and 2,4-D in flooded and nonflooded soils. Journal of Environmental Quality. 24 (3): 493-497.

Jones K. 1995. Triclopyr Butoxyethyl Ester: A Study of the Effect on Pregnancy of the Rat: Lab Project Number: DWC 644/932299: DWC 649/932299: DWC 644/649/932299. Unpublished study prepared by Huntingdon Research Centre Ltd. 277 p.

Kastl PE; Veenstra GE; Hermann EA. 1996. A pharmacokinetic comparison of triclopyr and its butoxyethyl ester in Fischer 344 rats. Unpublished study cited in Timchalk et al. 1990.

Kennard L et al. 2002. Risk Assessment in Support of a Study Entitled Degradation Path and Kinetics of Triclopyr Butoxyethyl Ester in Two European Water and Sediment Systems. (Study ID: 990057): Lab Project Number: LMK011802: 990057. Unpublished study prepared by Dow Agrosciences LLC. 15 p.

Kennard L; Smith K; Balcer J. 2001. Degradation Path and Kinetics of Triclopyr Butoxyethyl Ester in Two European Water and Sediment Systems: Lab Project Number: 990057. Unpublished study prepared by Dow Agrosciences LLC. 20 p.

Kirk HD; Hanley TH; Eisenbrandt DL; Quast JF. 1989. Evaluation of the potential developmental effects of triclopyr in the New Zealand white rabbit. Toxicologist. 9(1): 223.

Knisel WG; Davis FM. 1999. GLEAMS (Groundwater Loading Effects of Agricultural Management Systems), Version 3.0. User Manual. U.S. Department of Agriculture, Agricultural Research Service, Southeast Watershed Research Laboratory, Tifton, GA. Pub. No.: SEWRL-WGK/FMD-050199. Report Dated May 1, 1999 and revised August 15, 2000. 194pp.

Knisel WG; Davis FM; Leonard RA. 1992. GLEAMS Version 2.0 User Manual. U.S. Department of Agriculture, Agricultural Research Service, Southeast Watershed Research Laboratory, Tifton, GA. 202pp.

Knoefel PK; Huang KC. 1969. Renal tubular secretion and biosynthesis of organic acids. Proc Soc Exp Biol Med. 130(3): 914-919.

Knuteson J. 1999. Review of Environmental Fate of 3,5,6-Trichloro-2-pyridinol. (TCP): Laboratory, Terrestrial and Aquatic Field Studies: Lab Project Number: GH-C 4875. Unpublished study prepared by Dow AgroSciences LLC. 75 p.

Kreutzweiser DP; Holmes SB; Behmer DJ. 1992. Effects of the herbicides hexazinone and triclopyr ester on aquatic insects. Ecotoxicol Environ Saf. 23(3): 364-74.

Kreutzweiser DP; Holmes SB; Eichenberg DC. 1994. Influence of exposure duration on the toxicity of triclopyr ester to fish and aquatic insects. Archives of Environmental Contamination and Toxicology. 26(1): 124-129.

Kreutzweiser DP; Thompson DG; Capell SS; Thomas DR; Staznik B. 1995. Field evaluation of triclopyr ester toxicity to fish. Archives of Environmental Contamination and Toxicology. 28 (1): 18-26.

Kreutzweiser DP; Thompson DG; Staznik B; Shepherd JA. 1998. Accumulation dynamics of triclopyr ester in aquatic leaf packs and effects on detritivorous insects. Journal of Environmental Quality. 27 (5): 1138-1147.

Kuhn J. 2000a. Acute Dermal Toxicity Study in Rabbits: Triclopyr: Final Report: Lab Project Number: 5830-00. Unpublished study prepared by Stillmeadow, Inc. 13 p. {OPPTS 870.1200}.

Kuhn J. 2000b. Acute Eye Irritation Study in Rabbits: Triclopyr: Final Report: Lab Project Number: 5832-00. Unpublished study prepared by Stillmeadow, Inc. 18 p. {OPPTS 870.2400}.

Kuhn J. 2000c. Acute Dermal Irritation Study in Rabbits: Triclopyr: Final Report: Lab Project Number: 5833-00. Unpublished study prepared by Stillmeadow, Inc. 14 p. {OPPTS 870.2500}.

Kuhn J. 2000d. Skin Sensitization Study in Guinea Pigs: Triclopyr: Final Report: Lab Project Number: 5834-00. Unpublished study prepared by StillMeadow, Inc. 19 p. {OPPTS 870.2600}.

Kuhn J. 2001. Acute Oral Toxicity Study in Rats: Triclopyr: Final Report: Lab Project Number: 5829-00. Unpublished study prepared by Stillmeadow, Inc. 17 p. {OPPTS 870.1100}.

Lee CH; Oloffs PC; Szeto SY. 1986. Persistence degradation and movement of triclopyr and its ethylene glycol butyl ether ester in a forest soil. J Agric Food Chem. 34 (6): 1075-1079.

Leslie DM Jr; Soper RB; Lochmiller RL; Engle DM 1996. 1996. Habitat use by white-tailed deer on cross timbers rangeland following brush management. J Range Manag. 49(5): 401-406.

Leutenschlager RA; Bell FW; Wagner RG; Reynolds PE. 1998. The Fallingsnow Ecosystem Project: Documenting the consequences of conifer release alternatives. J Forest Ecology. 96(11): 10-27.

Lewer P; Owen WJ. 1990. Selective action of the herbicide triclopyr. Pestic Biochem Physiol. 36(2): 187-200.

Li ZC; Yost RS; Green RE. 1998. Incorporating uncertainty in a chemical leaching assessment. Journal of Contaminant Hydrology. 29 (4): 285-299.

Lickly TD; Murphy PG. 1987. The amount and identity of carbon-14 residues in bluegills lepomis-macrochirus exposed to carbon-14 triclopyr. Environ Int. 13 (2): 213-218.

Lindgren CJ; Gabor TS; Murkin HR. 1998. Impact of triclopyr amine on galerucella calmariensis l. (Coleoptera: chrysomelidae) and a step toward integrated management of purple loosestrife lythrum salicaria l. Biological Control. 12 (1): 14-19.

Lochmiller RL; Pietz DG; McMurry ST; Leslie DM; Engle DM. 1995. Alterations in condition of cottontail rabbits (Sylvilagus floridanus) on rangelands following brush management. J Range Manag. 48: 232-239.

Luken JO; Beiting SW; Kareth SK; Kumler RL; Liu JH; Seither CA. 1994. Target and nontarget discrimination of herbicides applied to vegetation in a power-line corridor. Environmental Management. 18 (2): 251-255.

Maddy KT; Edmiston S; Richmond D. 1990. Illness injuries and deaths from pesticide exposures in California USA 1949-1988. Reviews of Environmental Contamination and Toxicology. 114: 57-124.

Manclus JJ; Montoya A. 1996a. Development of an enzyme-linked immunosorbent assay for 3,5,6-trichloro-2-pyridinol. 1. production and characterization of monoclonal antibodies. Journal of Agricultural and Food Chemistry. 44 (11): 3703-3709.

Manclus JJ; Montoya A. 1996b. Development of an enzyme-linked immunosorbent assay for 3,5,6-trichloro-2-pyridinol. 2. assay optimization and application to environmental water samples. Journal of Agricultural and Food Chemistry. 44 (11): 3710-3716.

Marino T; Gilles M; Rick D et al. 1999. Evaluation of the Toxicity of 3,5,6-Trichloro-2-Pyridinol(TCP) to the Early Life Stages of the Rainbow Trout, Oncorhynchus mykiss Walbaum:. Lab Project Number: 991173. Unpublished study prepared by The Dow Chemical Co. 56 p.

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

Mayes MA; Dill DC; Bodner KM; Mendoza CG. 1984. Triclopyr triethylamine salt toxicity to life stages of the fathead minnow pimephales-promelas. Bull Environ Contam Toxicol. 33 (3): 339-347.

Mayes M. 1990a. Dow Chemical U S A Phase 3 Summary of MRID 00031251. One-generation Reproduction Study - Bobwhite Quail: Dowco 233; Final Report: Project ID: 103-191. Prepared by Wildlife International Ltd. 13 p.

Mayes M. 1990b. Dow Chemical U S A Phase 3 Summary of MRID 00031250. One-generation Reproduction Study - Mallard Duck Dowco 233; Final Report: Project ID: 103-174. Prepared by Wildlife International Ltd. 14 p.

Mayes M. 1990c. Dow Chemical U S A Phase 3 Summary of MRID 00151958. The Acute and Chronic Toxicity of Triclopyr [(3,5,6-Trichloro-2- Pyridiny)loxy) Acetic Acid] Triethylamine Salt Solution to the Fathead (Pimephales promelas Rafinesque): Minnow. Project ID: ES-582. Prepared by Dow Chemical CO. 11 p.

Mayes M. 1990d. Dow Chemical U S A Phase 3 Summary of MRID 00151959. The Acute and Chronic Toxicity of Triclopyr [[(3,5,6,- Trichloro-2- Pyridinyl)oxy) Acetic Acid] Triethylamine Salt Solution to Daphnia magna Straus:. Project ID: ES-583. Prepared by Dow Chemical CO. 10 p.

Mayes M. 1991a. Response to Phase 3 Submission on Triethylammonium Triclopyr.. [Avian Reproduction Test--Bobwhite!: Lab Project Number: GHRC 160. Unpublished study prepared by Dow Chemical Co., Environmental Tox & Chem Res. Lab. 4 p.

Mayes M. 1991b. Response to Phase 3 Submission on Triethylammonium Triclopyr.. [Avian Reproduction Test--Mallard!: Lab Project Number: GHRC 161. Unpublished study prepared by Dow Chemical Co., Environmental Tox & Chem Res. Lab. 39 p.

Mayes M. 1991c. Response to Phase 3 Submission on Triethylammonium Triclopyr..[Growth and Reproduction of Aquatic Plants Tier 2: Lemna gibba!: Lab Project Number: ES-970. Unpublished study prepared by Dow Chemical Co., Environmental Tox & Chem Res. Lab. 21 p.

Mayes M. 1991d. Response to Phase 3 Submission on Triethylammonium Triclopyr..[Growth and Reproduction of Aquatic Plants Tier 2: Skeletonema costatum!: Lab Project Number: ES-971. Unpublished study prepared by Dow Chemical Co., Environmental Tox & Chem. Res. Lab. 11 p.

Mayes M. 1991e. Response to Phase 3 Submission on Butoxyethyl Triclopyr..[Growth and Reproduction of Aquatic Plants Tier 2: Selenaestrum capricornutum!. Unpublished study prepared by Dow Chemical Co., Environmental Tox & Chem Res. Lab. 16 p.

McCall PJ; Gavit PD. 1986. Aqueous pH of triclopyr and its butoxyethyl ester and calculated environmental pH rates. Environ Toxicol Chem. 5 (10): 879-886.

McCall PJ; Laskowski DA; Bidlack HD. 1988. Simulation of the aquatic fate of triclopyr butoxyethyl ester and its predicted effects on Coho salmon. Environ Toxicol Chem. 7: 517-527.

McConnell, L.L. et al. 1998. Wet deposition of current use pesticides in the Sierra Nevada mountain range, California, USA. Environ Toxicol Chemi. 17:1908-1916.

McCormick R; Robb C. 2000. Dissipation of Dislodgeable Foliar Residues of Triclopyr from Treated Rice: Lab Project Number: 990023. Unpublished study prepared by Dow AgroSciences LLC. 119 p. {OPPTS 875.2100}.

McCormick R. 1994. Letter Sent to Office of Pesticide Programs Dated 4/28/94 concerning teratogenicity study on rats: Triclopyr Butoxyethyl Ester. Prepared by DowElanco. 2 p.

McKellar R; Norton E. 1977a. Residues of Triclopyr; 3, 5, 6-Tri- chloro-2-pyridinol and 2-Methoxy-3,5,6-Trichloropyridine in Soil and Water from a Watershed Treated with Garlon 3A Herbicide by Aerial Application: Laboratory ID: GH-C-989. Unpublished study prepared by Dow Chemical.

McKellar R; Norton E. 1977b. Residues of Triclopyr; 3,5,6 Tri- chloro-2-Pyridinol and 2-Methoxy-3, 5, 6-Trichloropyridine in Soil and Water Form [sic] a Watershed Treated with Garlon 3A Herbicide by Aerial Application: GH-C 989. Unpublished study prepared by Dow Chemical U.S.A. 50.

McMahon CK; Bush PB. 1992. Forest worker exposure to airborne herbicide residues in smoke from prescribed fires in the southern united states. Am Ind Hyg Assoc J. 53(4): 265-72.

McMaster S. 1997. Supplemental Information for Establishment of Residue Tolerances for the Herbicide Triclopyr in or on Fish and Shellfish and Allowable Residue Levels in Potable Water: Lab Project Number: SAM121697. Unpublished study prepared by DowElanco. 11 p.

McMurry ST; Lochmiller RL; Boggs JF; Leslie D M JR; Engle DM. 1993a. Woodrat population dynamics following modification of resource availability. Am Midl Nat. 129 (2): 248-256.

McMurry ST; Lochmiller RL; Boggs JF; Leslie DM; Engle DM. 1993b. Opportunistic foraging of eastern woodrats (Neotoma floridana) in manipulated habitats. Am Midland Natur. 130: 325-337.

Middendorf P. 1992. Forest Worker Exposures to Triclopyr, Butoxyethyl Ester Durking Streamline Basal Bark Applications of Garlon 4 Herbicide. Georgia Institute of Technology, Georgial Tech Research Institute, Atlanta, GA. Final Report Project #A-8112-000, 48 pp. plus appendices. Copy courtesy of Paul Mistretta, USDA/FS.

Middendorf P; Timchalk C; Kropscott B; Rick D. 1992. Forest worker exposures to triclopyr butoxyethyl ester during directed foliar applications of Garlon4. Proc South Weed Sci Soc. 45: 177-188.

Milazzo D; Batchelder T. 1981. Evaluation of Garlon 4 Formulation in the Aquatic Environment: Report No. ES-426. Unpublished study prepared by Dow Chemical U.S.A. 21 p.

Mineau P; Boersma DC; Collins B. 1994. An analysis of avian reproduction studies submitted for pesticide registration. Ecotoxicology and Environmental Safety. 29 (3): 304-329.

Mizell M ; Lomax L. 1988. Garlon 3A (Triclopyr as Triethylamine Salt): Acute Oral Toxicity Study in Fischer 344 Rats: Lab Project Number: M-003724-009A. Unpublished study prepared by The Dow Chemical Co. 28 p. (Triclopyr as Triethylamine Salt): Acute Oral Toxicity Study in Fischer 344 Rats: Lab Proj- ect Number: M-003724-009A. Unpublished study prepared by The Dow Chemical Co. 28 p.

Mizell M; Lomax L. 1989. Garlon 3A (Triclopyr as Triethylamine Salt): Acute Dermal Toxicity Study in New Zealand White Rabbits: Lab Project Number: M-003724-009D. Unpublished study prepared by The Dow Chemical Co. 20 p. (Triclopyr as Triethylamine Salt): Acute Dermal Toxicity Study in New Zealand White Rabbits: Lab Project Number: M-003724-009D. Unpublished study prepared by The Dow Chemical Co. 20 p.

Mizell M. 1988a. Garlon 3A (Triclopyr as Triethylamine Salt): Primary Eye Irritation Study in New Zealand White Rabbits: Lab Project Number: M-003724-009C. Unpublished study prepared by The Dow Chemical Co. 14 p. (Triclopyr as Triethylamine Salt): Primary Eye Irritation Study in New Zealand White Rabbits: Lab Project Number: M-003724-009C. Unpublished study prepared by The Dow Chemical Co. 14 p.

Mizell M. 1988b. Garlon 3A (Triclopyr as Triethylamine Salt): Primary Dermal Irritation Study in New Zealand White Rabbits: Lab Project No. M-003724-009B. Unpublished study prepared by the Dow Chemical Co. 12 p. (Triclopyr as Triethylamine Salt): Primary Dermal Irritation Study in New Zealand White Rabbits: Lab Project No. M-003724-009B. Unpublished study prepared by the Dow Chemical Co. 12 p.

Mizell M. 1989. Garlon 3A (Triclopyr as Triethylamine Salt): Dermal Sensitization Potential in the Hartley Albino Guinea Pig: Lab Project Number: M-003724-009E: DR-0291-2256-005E1. Unpub- lished study prepared by The Dow Chemical Co. 14 p. (Triclopyr as Triethylamine Salt): Dermal Sensitization Potential in the Hartley Albino Guinea Pig: Lab Project Number: M-003724-009E: DR-0291-2256-005E1. Unpub- lished study prepared by The Dow Chemical Co. 14 p.

Moore JA. 1964. Physiology of the Amphibia. Academic Press, New York. 654 p. (Cited in USDA 1993).

Morash R; Freedman B. 1989. The effects of several herbicides on the germination of seeds in the forest floor. Can J for Res. 19 (3): 347-350.

Morgan JD; Vigers GA; Farrell AP; Janz DM; Manville JF. 1991. Acute avoidance reactions and behavioral responses of juvenile rainbow trout (Oncorhynchus mykiss) to Garlon 4, Garlon 3a and vision herbicides. Environ Toxicol Chem. 10(1): 73-80.

Morre DJ; Morre JT; Lawrence J; Moini M. 1998. Activity of triclopyr herbicide enhanced by combination with cobalt chloride or ammonium nitrate. Journal of Plant Growth Regulation. 17 (3): 125-129.

Moslen MT. 1996. Toxic Responses of the Liver. In: Casarett and Doull's Toxicology: The Basic Science of Poisons. 5th Edition. McGraw-Hill, Health Professions Division, New York, NY. pp. 403-415.

NPIRS (National Pesticide Information Regrieval System). 2002. On line data base provided by the Center for Environmental and Regulatory Information Systems at Purdue University.

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

NRC (National Research Council). 1993. Pesticides in the Diets of Infants and Children. Committee on Pesticides in the Diets of Infants and Children. National Academy Press, Washington, DC.

Neary DG; Michael JL. 1996. Herbicides. protecting long-term sustainability and water quality in forest ecosystems. New Zealand Journal of Forestry Science. 26(1-2): 241-264.

Neary DG; Bush PB; Mcmahon CK; Cantrell RL; Taylor J W JR. 1988. Persistence of nine forest pesticides in the surface horizon of a typic quartzipsamment soil of the Ocala national forest Florida USA. Soil Crop Sci Soc Fla Proc. 47: 127-134.

Neary DG; Bush PB; Michael JL. 1993. Fate dissipation and environmental effects of pesticides in southern forest a review of a decade of research progress. Environ Toxicol Chem; 12 (3) 1993 411-428.

Netherland MD; Getsinger KD. 1992. Efficacy of triclopyr on eurasian watermilfoil: concentration and exposure time effects. J Aquat Plant Manage; 30 (0) 1992 1-5.

Newmaster SG; Bell FW; Vitt DH. 1999. The effects of glyphosate and triclopyr on common bryophytes and lichens in northwestern Ontario. Canadian Journal of Forest Research. 29 (7): 1101-1111.

Newton M; Roberts F; Allen A; Kelpsas B; White D; Boyd P. 1990. Deposition and dissipation of three herbicides in foliage, litter, and soil of brushfields of southwest Oregon (USA). J Agric Food Chem. 38 (2): 574-583.

Nigg HN. 1998. Occupational Monitoring. Chapter 5 in: Occupational Hazards of Pesticide Exposure - Sampling, Monitoring, Measuring. Taylor and Francis Press. Philadelphia PA. pp. 107-134.

Nolte KR; Fulbright TE. 1997. Plant, small mammal and avian diversity following control of honey mesquite. J Range Manag. 50(2): 205-212.

Norris LA; Montgomery ML; Warren LE. 1987. Triclopyr persistence in western Oregon hill pastures. Bull Environ Contam Toxicol. 39(1):134-41.

Nugent P; Schotts B. 1990. Determination of Residues in a Forest Ecosystem Resulting from the Aerial Application of Garlon 4 Herbicide: Lab Project Number: GH/C/2283. Unpublished study prepared by DowElanco. 123 p.

O'Flaherty EJ. 1981. Toxicants and Drugs: Kinetics and Dynamics. John Wiley and Sons, New York, NY. 398 pp.

Obenshain KR; Metcalf MC; Abdelghani AA; Regnes JL; Hodges DG; Swalm CM. 1997. Spatial analysis of herbicide decay rates in louisiana. Environmental Monitoring and Assessment. 48 (3): 307-316.

Osweiler GD. 1983. Toxicology of triclopyr herbicide in the equine. Am Assoc Vet Lab Diagnost. 26th Ann Proc. pp. 193-201.

Pell M; Stenberg B; Torstensson L. 1998. Potential denitrification and nitrification tests for evaluation of pesticide effects in soil. Ambio. 27 (1): 24-28.

Perkins PJ; Boermans HJ; Stephenson GR. 2000. Toxicity of glyphosate and triclopyr using the frog embryo teratogenesis assay--xenopus. Environ Toxicol Chem. 19(4): 940-5.

Peterson HG; Boutin C; Martin PA; Freemark KE; Ruecker NJ; Moody MJ. 1994. Aquatic phytotoxicity of 23 pesticides applied at expected environmental concentrations. Aquatic Toxicology (Amsterdam). 28(3-4): 275-292.

Peterson JL; Jepson PC; Jenkins JJ. 2001. A test system to evaluate the susceptibility of Oregon, AUS, native stream invertebrataes to triclopyr and carbaryl. Environ Toxicol Chem. 20(10): 2205-14.

Peterson J. 2000. Additional Historical Control Data for MRID 43675801--Triclopyr Butoxyethyl Ester: a Study of the Effect on Pregnancy of the Rat: Lab Project Number: JRP07202000: DWC644. Unpublished study prepared by Dow AgroSciences LLC. 6 p.

Petty DG; Skogerboe JG; Getsinger KD; Foster DR; Fairchild JW. 1998. Aquatic dissipation of triclopyr in a whole-pond treatment. NTIS/ADA356971.

Petty DG; Skogerboe JG; Getsinger KD; Foster DR; Houtman BA; Fairchild JF; Anderson LW. 2001. The aquatic fate of triclopyr in WHOLE-POND treatments. Pest Manag Sci. 57(9): 764-75.

Petty D. 2002. Potential Triclopyr Use in Commercially Fished Non-Flowing Waters of the United States: Lab Project Number: NDR200201. Unpublished study prepared by NDR Research. 20 p.

Phillips A; Ervick D. 1991. Determination of Residues of Triclopyr and 3,5,6-Trichloro-2-pyrinidol in Apples and Apple Process Fractions Following Applications of Garlon 3A Herbicide to Apple Orchard Floors: Lab Project Number: GHC-2528: 88096. Unpublished study prepared by DowElan.

Piper WN; Rose JQ; Leng ML; Gehring PJ. 1973. The fate of 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) following oral administration to rats and dogs. Toxicol. Appl. Pharmacol. 26: 339-351.

Plaumann D; Mackasey M; Oostenbrink P. 1983a. Residues of Triclopyr (Garlon 4) in Soil when Applied by Ground to a Right-of-way: Report No. GHS-C-54. Unpublished study prepared by Dow Chemical Canada Inc. 17 p. (Garlon 4) in Soil when Applied by Ground to a Right- of-way: Report No. GHS-C-54. Unpublished study prepared by Dow Chemical Canada Inc. 17 p.

Plaumann D; Mackasey M; Oostenbrink P. 1983b. Residues of Triclopyr (Garlon 4) in Soil When Aerialy Applied to a Forest Ecosystem: GHS-C-53. Unpublished study prepared by Dow Chemical Canada Inc. 26 p. (Garlon 4) in Soil When Aerialy Applied to a Forest Eco- system: GHS-C-53. Unpublished study prepared by Dow Chemical Canada Inc. 26 p.

Poletika N; Phillips A. 1996. Field Dissipation of Triclopyr in Southern U.S. Rice Culture: Lab Project Number: ENV94015. Unpublished study prepared by A&L Great Lakes Laboratories, Inc.; North American Environmental Chemistry Laboratory, DowElanco; and Mid-South Weed Scientists, In.

Powers RF; Ferrell GT. 1996. Moisture, nutrient, and insect constraints on plantation growth: the "garden of eden" study. New Zealand Journal of Forestry Science. 26 (1-2): 126-144.

Pruitt P. 1998. Determination of Physical and Chemical Properties of Pathfinder II(NAF-5),. A Liquid End-Use Product Containing Triclopyr-2-butoxyethyl Ester: Lab Project Number: FOR97114. Unpublished study prepared by Dow AgroSciences LLC. 9 p. {OPPTS 830.6316}.

Pusino A; Liu W; Gessa C. 1994. Adsorption of triclopyr on soil and some of its components. Journal of Agricultural and Food Chemistry. 42 (4): 1026-1029.

Quast JF; Humiston CG; LeBeau JE; et al. 1976. 3,5,6- Trichloro-2-pyridyloxyacetic Acid (Dowco^(TM) 233 Herbicide): Subchronic Dietary Feeding Study in Beagle Dogs. (Unpublished study received May 1, 1981 under 464-554; submitted by Dow Chem- ical U.S.A., Midland, Mich.; CDL:070043-C).

Quast JF; Wade CE; Kalnins RV; et al. 1977. 3,5,6-Tri- chloro-2-pyridinyloxyacetic Acid (Dowco^(TM) 233 Herbicide): Supplemental Subchronic Dietary Feeding Study in Beagle Dogs. (Unpublished study received May 1, 1981 under 464-554; submitted by Dow Chemical U.S.A., Midland, Mich.; CDL:070043-D).

Quast J; Gushow T; Stott W; et al. 1988. Triclopyr: A One Year Dietary Toxicity Study in Beagle Dogs: Project Study ID's: K-042085-036; K-042085-36F. Unpublished study prepared by Dow Chemical Co. 216 p.

Quast J. 1990. Dow Chemical U S A Phase 3 Summary of MRID 41200301. Triclopyr: A One Year Dietary Toxicity Study in Beagle Dogs: Project ID: HET K-042085-036. Prepared by Dow Chemical CO. 9 p.

Rawn D FK; Halldorson T HJ; Woychuk RN; Muir D CG. 1999. Pesticides in the red river and its tributaries in southern manitoba: 1993-95. Water Quality Research Journal of Canada. 34(2): 183-219.

Reeves GL. 1995. The mobility of triclopyr under laboratory and field conditions. Walker, A, et al.(ed) British Crop Protection Council Monograph, No 62 Pesticide Movement to Water; Symposium, Coventry, England, Uk, April 3-5, 1995 Xiv+414p British Crop Protection Council: Farnham, England, UK ISBN 0-948404-85-x.

Reid CP; Hurtt W. 1970. Root exudation of herbicides by woody plants: Alleolopathic implication. Nature. 225: 291.

Rick D; Kirk H; Fontaine D; et al. 1996. The Nature of Triclopyr Residues in the Bluegill, Lepomis macrochirus Rafinesque: Lab Project Number: DECO-ES-2761. Unpublished study prepared by The Dow Chemical Co. 64 p.

Ritter A; Peacock A. 2000. Aquatic Dissipation Modeling of Triclopyr: Lab Project Number: WEI 369.06: 30398118-5023-1. Unpublished study prepared by Waterborne Environmental, Inc. 56 p.

Roshon RD; Mccann JH; Thompson DG; Stephenson GR. 1999. Effects of seven forestry management herbicides on myriophyllum sibiricum, as compared with other nontarget aquatic organisms. Canadian Journal of Forest Research. 29(7):1158-1169.

Ross D; Pell I. 1981. The Acute Toxicity of Garlon 4E to Rain- bow Trout (..): Report No. DWC 336. (a)/81750. Unpublished study prepared by Huntingdon Research Centre. 15 p.

Rowe LD; Cysewski SJ; Palmer SJ. 1980. Acute Experimental Triclopyr Poisoning in Cattle. Unpublished report presented at the Scientific Session of the American College of Veterinary Toxicologists. July 21, 1980, Washington, DC. (Cited in SERA 1996).

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

SERA (Syracuse Environmental Research Associates, Inc.). 1996. Selected Commercial Formulations of Triclopyr - Garlon 3A and Garlon 4 Risk Assessment. SERA TR 95-22-02-02a, report dated March 31, 1996.

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

Sar TK; Bagchi B; Das SK; Mandal TK; Chakraborty AK; Bhattacharyya A; Choudhury A. 2002. Toxicokinetics, recovery, and metabolism of triclopyr butotyl (ACTP) ester in goats. J Agric Food Chem. 50(15): 4202-4209.

Sassaman JF; Pienta R; Jacobs M; Cioffi J. 1984. Pesticide background statements. Volume 1. herbicides. Govt Reports Announcements & Index, Issue 24, 1989.

Schardein J; Scialli A. 2001. Triclopyr: Review of Developmental and Reproductive Studies and Rebuttal of EPA HED Documents 014472 and 014395: Lab Project Number: GHC-5356. Unpublished study prepared by Dow AgroSciences LLC. 93 p.

Schulz CA; Leslie D M JR; Lochmiller RL; Engle DM. 1992a. Autumn and winter bird populations in herbicide-treated cross timbers in oklahoma. Am Midl Nat. 127 (2): 215-223.

Schulz CA; Leslie DM; Lochmiller RL; Engle DM. 1992b. Herbicide effects on cross timbers breeding birds. J Range Manag. 45: 407-411.

Schwab D. 1995. Evaluating the Effects of Triclopyr BEE on the Seedling Emergence and Vegetative Vigor of Non-Target Terrestrial Plants: Lab Project Numbers: 41964: RES94090. Unpublished study prepared by ABC Labs., Inc. 152 p.

SePRO Corporation. 2003a. Renovate 3 Specimen Label. Form No. A-00-RG-01(03)SePRO Corporation, Carmel, IN.

SePRO Corporation. 2003b. Renovate 3 Material Safety Data Sheet. Form No. A-02-RG-01(03)SePRO Corporation, Carmel, IN.

Segawa R; Bradley A; Lee P; Tran D; Hsu J; White J; Goh KS. 1997. Residues of forestry herbicides in plants of importance to California native americans. Bulletin of Environmental Contamination and Toxicology. 59 (4): 556-563.

Seiler JR; Cazell BH; Schneider WG; Zedaker SM; Kreh RE. 1993. Effect of plant moisture stress on absorption and translocation of triclopyr in oak seedlings. Canadian Journal of Forest Research. 23 (10): 2213-2215.

Servizi JA; Gordon RW; Martens DW. 1987. Acute toxicity of Garlon 4 and Roundup herbicides to salmon, daphnia, and trout. Bull Environ Contam Toxicol. 39: 15-22.

Shackelford DD; Young DL; Mihaliak CA; Shurdut BA; Itak JA. 1999. Practical immunochemical method for determination of 3,5, 6-trichloro-2-pyridinol in human urine: applications and considerations for exposure assessment. J Agric Food Chem. 47(1):177-82.

Sidhu SS: Chakravarty P. 1990. Effect of selected forestry herbicides on ectomycorrhizal development and seedling growth of lodgepole pine and white spruce under controlled and field environment. European Journal of Forest Pathology. 20:77-94.

Siltanen H; Rosenberg C; Raatikainen M; Raatikainen T. 1981. Triclopyr, glyphosate and pH residues in cowberries, bilberries and lichen. Bull Environ Contam Toxicol. 27(5):731-7.

Smith CT; McCormack ML. 1988. Watershed losses of triclopyr after aerial application to release spruce-fir. Proc Ann Meet Northeast Weed Sci. 42: 104-108.

Smith FA; Schwetz BA; Nitschke KD; et al. 1960?. The Effect of Dowco 233 Herbicide (3,5,6-Trichloro-2-pyridyloxyacetic acid) on the Developing Embryo and Fetus of Pregnant Rabbits. (Unpublished study received May 10, 1977 under 464-546; submit- ted by Dow Chemical U.S.A., Midland, Mich.; CDL:229780-E).

Snipes CE; Street JE; Mueller TC. 1991. Cotton (Gossypium hirsutum) response to simulated triclopyr drift. Weed Technol. 5(3): 493-498.

Solomon KR; Bowhey CS; Liber K; Stephenson GR. 1988. Persistence of Hexazinone (Velpar), Triclopyr (Garlon) and 2,4-D in a northern Ontario Canada aquatic environment. J Agric Food Chem. 36(6): 1314-1318.

Spencer JR. 2000. Letter to Dave Bakke concernig Spencer et al. 2000 exposure study of hand applications to triclopyr. Copy courtesy of Dave Bakke, USDA/FS/R5.

Spencer JR; Edmiston S; Cowan C; Hernandex BZ; Schneider F; Quan V. 2000. Exposure of hand applicators to triclopyr in forest settings, 1995. California EPA, Dept of Pesticide Regulation, Worker Health and safety Branch. Health and safety Report HS-1769. February 29, 2000. 47 pages.

Sprecher SL; Stewart AB. 1995. Triclopyr effects on peroxidase activity in target and non-target aquatic plants. Journal of Aquatic Plant Management. 33 : 43-48.

Stephenson GR; Solomon KR; Bowhey CS; Liber K. 1990. Persistence, leachability, and lateral movement of triclopyr (Garlon) in selected canadian forestry soils. J Agric Food Chem. 38 (2): 584-588.

Street JE; Baldwin JL; Mueller TC. 1992. Influence of growth stage on rice (Oryza sativa) tolerance to triclopyr. Weed Technol. 6(4): 930-933.

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

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

Stritzke JF; Engle DM; Mccollum FT. 1991. Vegetation management in the cross timbers: response of woody species to herbicides and burning. Weed Technol; 5 (2): 400-405.

Swadener C. 1993. Triclopyr. J Pest Reform. 13: 29-35.

Teske ME; Curbishley TB. 1990. Forest Service Aerial Spray Computer Model, FSCBF 4.0 User Manual. Continuum Dynamics, Inc, Princeton, NJ. CDI Report No. 90-06.

Teske ME; Bird SL; Esterly DM; Ray SL; Perry SG. 2001. A User's Guide for AgDRIFT 2.0: A Tiered Approach for the Assessment of Spray Drift. Continuum Dynamics, Inc. Public Use Version. C.D.I. Report No. 01-01. Available, with executable model at: http://www.agdrift.com/.

Texas Dept of Agriculture. 1992. Report of an Investigation Concerning Alleged Adverse Effects of Garlon 3A and Rodeo on Horses and Property: Lab Project Number: 10-91-0075. Unpublished study. 31 p.

Tharr D. 1994. Forest worker exposure to Garlon 4 herbicide. Applied Occupational and Environmental Hygiene. 9(9): 589-594.

Thompson DJ; Dyke IL; Lower CE; et al. 1979. Results of a Teratology Study on Dowco 233 (3,5,6-Trichloro-2-pyridyloxy) Acetic Acid in the Rat. (Unpublished study received May 1, 1981 under 464-554; submitted by Dow Chemical U.S.A., Midland, Mich.; CDL:070044-B).

Thompson DG; Staznik B; Fontaine DD; Mackay T; Oliver GR; Troth J. 1991. Fate of triclopyr ester (Release) in a boreal forest stream. Environ Toxicol Chem. 10 (5): 619-632.

Thompson DG; Kreutzweiser DP; Capell SS; Thomas DR; Staznik B; Viinikka T. 1995. Fate and effects of triclopyr ester in a first-order forest stream. Environ Toxicol Chem. 14 (8): 1307-1317.

Thompson, D.G., et al. 2000. Comparative fate of glyphosate and triclopyr herbicides in the forest floor and mineral soil of an Acadian forest regeneration site. Canadian Journal of Forest Research. 30:1808-1816.

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

Timchalk C; Dryzga MD; Kastl PE. 1990. Pharmacokinetics and metabolism of triclopyr 3,5,6-trichloro-2-pyridinyloxyacetic acid) in fischer 344 rats. Toxicology. 62(1): 71-87.

Timchalk C; Lacher J; Dryzga M; et al. 1995. Triclopyr (3,5,6-Trichloro-2-pyridinyloxyacetic acid): Pharmacokinetics in the Dog: Lab Project Number: K-42085-(49). Unpublished study prepared by The Dow Chemical Co. 99 p.

Timchalk C; Finco DR; Quast JF. 1997. Evaluation of renal function in rhesus monkeys and comparison to beagle dogs following oral administration of the organic acid triclopyr (3,5,6-trichloro-2-pyridinyloxyacetic acid). Fundam Appl Toxicol. 36(1): 47-53.

Tsuda S; Ebino K; Ikeda M; et al. 1987. Triclopyr: 22-month Oral Chronic Toxicity and Oncogenicity Study in Mice. Unpub- lished study prepared by The Institute of Environmental Toxicol- ogy. 1465 p.

Turner EG; Getsinger KD; Netherland MD. 1994. Correlation of triclopyr and rhodamine wt dye dissipation in the pend oreille river. Journal of Aquatic Plant Management. 32 : 39-41.

U.S. EPA/OPP. 1995. RfD/Peer Review Report of Triclopyr, dated Jan 12, 1995. Copy courtesy of Janet Bressant, Public Information and Records Integrity Branch, May 25, 2001.

U.S. EPA/OPP (U.S. Environmental Protection Agency/Office of Pesticide Programs). 1998a. Reregistration Eligibility Decision (RED): triclopyr. www.epa.gov.

U.S. EPA/OPP(U.S. Environmental Protection Agency/Office of Pesticide Programs). 1998b. Human Health Assessment for Triclopyr Reregistration Eligibility Decision Document (RED). Copy courtesy of Janet Bressant, Public Information and Records Integrity Branch, Aug. 14, 2002.

(U.S. Environmental Protection Agency/Office of Pesticide Programs). 1998c. Environmental Fate and Effects Branch Chapter for Triclopyr Reregistration Eligibility Decision Document (RED). Copy courtesy of Janet Bressant, Public Information and Records Integrity Branch, Aug. 14, 2002.

U.S. EPA/OPP (U.S. Environmental Protection Agency/Office of Pesticide Programs). 2001. Lists of Inert Pesticide Ingredients. Http://www.epa.gov/opprd001/inerts/. Updated June 12, 2001.

U.S. EPA/OPP (U.S. Environmental Protection Agency/Office of Pesticide Programs). 2002. EPA: Federal Register: Triclopyr; Pesticide Tolerance. Federal Register. 67(181): 58712-58725.

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

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

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

U.S. EPA/ORD (U.S. Environmental Protection Agency/Office of Research and Development). 1993. Wildlife Exposure Factors Handbook. Volumes 1 and 2. EPA/600/R-93/187a,b. Pagination not continuous. Available NTIS: PB94-174778 and PB94-174779.

U.S. EPA/ORD (U.S. Environmental Protection Agency/Office of Research and Development). 1996. Exposure Factors Handbook. National Center for Environmental Assessment, U.S. EPA, Washington, DC. EPA/600/P-95/002Ba-c. Avail. NTIS: PB97-117683, 97-117691, PB97-117709.

USDA/APHIS (U.S. Department of Agriculture Animal and Plant Health Inspection Service). 1993. Nontarget Risk Assessment for the MEDFLY Cooperative Eradication Program. USDA Animal and Plant Health Inspection Service. February 1993.

USDA/ARS (U.S. Department of Agriculture, Agricultural Research Station). 1995. Pesticide Properties Database. http://wizard.arsusda.gov/acsl/ppdb3.html.

USDA/FS (U.S. Department of Agriculture/Forest Service). 2002. Regional Reports of Pesticide Use on National Forest System Lands: Fiscal Year 2001. Available at: www.fs.fed.us/foresthealth/pesticide/pur/reports.html.

USGS. 1998. USGS Annual Use Maps for Pesticides for 1992. Revised Oct. 23, 1998. http://wwwdwatcm.wr.usgs.gov/pnsp/use92.html.

Vandervoort C; Zabik MJ; Branham B; Lickfeldt DW. 1997. Fate of selected pesticides applied to turfgrass effect of composting on residues. Bulletin of Environmental Contamination and Toxicology. 58(1): 38-45.

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

Vedula U; Breslin W; Kropscott B; et al. 1995. Triclopyr: Two-Generation Dietary Reproduction Study in Sprague-Dawley Rats: Lab Project Number: K-042085-048: K-042085-048P1: K-042085-048G0. Unpublished study prepared by Dow Chemical Co. 1065 p.

Vogel A. 1998. Methodology and determination of 2 4-D and Triclopyr residues employing the gc-itd in the analysis of lettuce plants cultivated in the tala valley republic of south africa. Bulletin of Environmental Contamination and Toxicology. 60(3): 371-378.

WHO (World Health Organization). 1988. IARC Monographs on the Evaluation of Carcinogenic Risk to Humans: Alcohol Drinking. IARC Working Group on the Evaluation of Carcinogenic Risks to Humans, Lyon, France. International Agency for Research on Cancer, World Health Organization, Geneva Switzerland. pp. 122-125.

Wahlers RL; Burton JD; Maness EP; Skroch WA. 1997. Physiological characteristics of stem cut and blade delivery method of application. Weed Science. 45 (6): 746-749.

Wan MT; Moul DJ; Watts RG. 1987. Acute toxicity to juvenile pacific salmonids of Garlon 3a Garlon 4 triclopyr triclopyr ester and their transformation products 3 5 6 trichloro-2-pyridinol and 2 methoxy-3 5 6-trichloropyridine. Bull Environ Contam Toxicol. 39(4): 721-728.

Wan MT; Watts RG; Moul DJ. 1989. Effects of different dilution water types on the acute toxicity to juvenile Pacific salmonids and rainbow trout of glyphosate and its formulated products. Bull Environ Contam Toxicol. 43(3): 378-385.

Wan MT; Watts RG; Moul DJ. 1991. Acute toxicity to juvenile pacific northwest salmonids of basacid blue nb755 and its mixture with formulated products of 2 4-d glyphosate and triclopyr. Bull Environ Contam Toxicol. 47(3): 471-478.

Ward T; Boeri R. 1991a. Garlon 4 Herbicide: Acute Flow-through Toxicity to the Tidewater Silverside, Menidia beryllina: Lab Project Number: ES-DR-0224-6186-6. Unpublished study prepared by Resource Analysts, Inc. 26 p.

Ward T; Boeri R. 1991b. Garlon 4 Herbicide: Acute Flow-through Toxicity to Grass Shrimp, pugio: Lab Project No: ES-DR-0224- 6186. Unpublished study prepared by Resource Analysts, Inc. 27 p.

Ward T; Boeri R. 1991c. Garlon 4 Herbicide: Acute Flow-through Shell Deposition Test with the Eastern Oyster, Crassostrea virginica: Lab Project Number: ES-DR-0224-6186-7. Unpublished study prepared by Resource Analysts, Inc. 26 p.

Watanabe E; Hoshino R; Kanzaki Y; Tokumoto H; Kubo H; Nakazawa H. 2002. New approach to immunochemical determinations for triclopyr and 3,5,6-trichloro-2-pyridinol by using a bifunctional hapten, and evaluation of polyclonal antiserum. J Agric Food Chem. 50(13): 3637-46.

Webb WI; Newton M. 1972. Release of Picloram from roots. Weed Res. 12: 391-394.

Weber H. 2001. Group A-Product Chemistry Analysis of Triclopyr Butoxyethyl Ester Group A-Product Identity, Composition, and Analysis: Final Report: Lab Project Number: 310045.1.036.01. Unpublished study prepared by Midwest Research Institute. 88 p. {OPPTS 830.1700, 8.

Weber H. 2001. Group B-Physical and Chemical Properties Testing of Triclopyr Butoxyethyl Ester: Final Report: Lab Project Number: 310045.1.036.02. Unpublished study prepared by Midwest Research Institute. 48 p. {OPPTS 830.6302, 830.6303, 830.6304, 830.6313, 830.7000,.

Weinberg J; Hugo J; Miller J. 1994a. Evaluation of the Acute Toxicity of Garlon 4 Herbicide to the Bluegill, Lepomis macrochirus Rafinesque: Lab Project Number: DECO-ES-2854. Unpublished study prepared by The Dow Chemical Co. 25 p.

Weinberg J; Hugo J; Miller J. 1994b. Evaluation of the Acute Toxicity of Garlon 4 Herbicide to the Rainbow Trout, Oncorhynchus mykiss Walbaum: Lab Project Number: DECO-ES-2853. Unpublished study prepared by The Dow Chemical Co. 25 p.

Weinberg J; Hugo J; Massaro L et al. 1994c. Evaluation of the Acute Toxicity of Garlon 4 Herbicide to the Daphnid, Daphnia magna Straus: Lab Project Number: DECO-ES-2855. Unpublished study prepared by The Dow Chemical Co. 25 p.

Whisenant SG; Mcarthur ED. 1989. Triclopyr persistence in northern idaho usa forest vegetation. Bull Environ Contam Toxicol. 42(5): 660-665.

Wilcock RJ; Costley KJ; Cowles RJ; Wilson B; Southgate P. 1991. Stream run-off losses and soil and grass residues of triclopyr applied to hillside gorse. N Z J Agric Res. 34(3): 351-358.

Wilson BW; Sanborn JR; O'malley MA; Henderson JD; Billitti JR. 1997. Monitoring the pesticide-exposed worker. Occupational Medicine (Philadelphia). 12(2): 347-363.

Winegardner DL. 1996. An Introduction to Soils for Environmental Professionals. CRC Press, Boca Raton, Florida. 270 pp.

Wisniewski DF; Rothman LD. 1978. Analysis of Garlon 3A Herbicide for Possible Presence of N-Nitrosodiethylamine: ML-AL 78- 00054. (Unpublished study received Sep 26, 1978 under 464-546; submitted by Dow Chemical U.S.A., Midland, Mich.; CDL:235246-B).

Wolt J; Weglarz T; Wright J; et al. 1997. Triclopyr Non-Target Plant Risk Assessment: Lab Project Number: GH-C 4357. Unpublished study prepared by DowElanco. 95 p.

Wolt J. 1995. Anaerobic Aquatic Metabolism of. (carbon 14)-Triethylamine: Lab Project Number: ENV94086. Unpublished study prepared by DowElanco's North American Environmental Chemistry Lab. 102 p.

Wolt J. 1997. Ground and Surface Water Exposure Assessment for Triclopyr: Lab Project Number: GH-C 4350. Unpublished study prepared by DowElanco. 70 p.

Woodburn KB; Batzer FR; White FH; Schultz MR. 1993a. The aqueous photolysis of triclopyr. Environ Toxicol Chem. 12: 43-55.

Woodburn KB; Green WR; Westerdahl HE. 1993b. Aquatic dissipation of triclopyr in Lake Seminole, Georgia. Journal of Agricultural and Food Chemistry. 41(11): 2172-2177.

Worrall F; Wooff DA; Seheult AH; Coolen F PA. 1998. A bayesian approach to the analysis of environmental fate and behaviour data for pesticide registration. Pesticide Science. 54(2): 99-112.

Yackovich P; Lardie T. 1995a. Greenhouse Study to Determine the Nature of the Residue of Triclopyr Applied Foliarly to Radishes: Lab Project Number: MET93098. Unpublished study prepared by DowElanco North American Environmental Chemistry Lab. 111 p.

Yackovich P; Lardie T. 1995b. A Nature of the Residue Study in Apples from Soil Applied (carbon 14)-Triclopyr:. Lab Project Number: MET94070. Unpublished study prepared by DowElanco North American Environmental Chemistry Lab. 64 p.

Yackovich P; Lardie T. 1996c. A Nature of the Residue Study of (carbon 14)-Triclopyr Applied Foliarly in Irrigation Water to Apples: Lab Project Number: MET94077. Unpublished study prepared by DowElanco North American Environmental Chemistry Lab and Plant Sciences, Inc. 106 p. (carbon 14)-Triclopyr Applied Foliarly in Irrigation Water to Apples: Lab Project Number: MET94077. Unpublished study prepared by DowElanco North American Environmental Chemistry Lab and Plant Sciences, Inc. 106 p.

Yackovich P; Lardie T. 1996d. A Probe Study to Determine the Nature of the Residues of (carbon 14)-Labeled Triclopyr Applied to Apple Leaves and Fruit:. Lab Project Number: MET93110. Unpublished study prepared by DowElanco North American Environmental Chemistry Lab. 84 p.

Zahm SH. 1997. Mortality study of pesticide applicators and other employees of a lawn care service company. Journal of Occupational and Environmental Medicine; 39 (11) 1997 1055-1073.

APPENDICES

Appendix 1: Triclopyr Formulations with Forestry Applications (C&P Press 2002)		
Brand Name Company/Composition	**Application Rate (Specified by Label)**	**Inerts (Specified)**
Forestry Garlon 4 ***Specialty Herbicide*** **DOW AGROSCIENCES** ***Maximum Application Rate: 8 quarts/acre*** 61.6% triclopyr (**butoxy ethyl ester**) 38.4% inert ingredients 4 lbs a.e./gallon liquid formulation **Recommends use of surfactant for best results with all spray mixtures; an agricultural surfactant should be used at a rate of 1 to 2 quarts/acre** ***Aerial applications by helicopter ONLY***	**FOLIAR APPLICATIONS:** 1 to 8 qt/acre to control broadleaf weeds and woody plants	38.4% total other ingredients including: kerosene CAS No. 8008-20-6 proprietary surfactants contains petroleum distillates
	HIGH VOLUME FOLIAR TREATMENT WITH GROUND EQUIPMENT:: 1 to 3 qt/100 gallons of water; apply at a volume of 100 to 400 gallons of total spray/acre, depending on size and density of woody plants **LOW VOLUME FOLIAR TREATMENT WITH GROUND EQUIPMENT:** mix up to 20 qts in 10 to 100 gallons of finished spray	
	BROADCAST APPLICATION WITH GROUND EQUIPMENT:: **WOODY PLANT CONTROL**: **FOLIAGE TREATMENT**: Use 4 to 8 quarts in enough water to make 5 or more gallons/acre of total spray **BROADLEAF WEED CONTROL**: 1 to 4 quarts in a total volume of 5 or more gallons/acre as a water spray mixture	
	FOREST MANAGEMENT APPLICATIONS: 5 to 25 gallons/acre by air or 10 to 100 gallons/acre by ground	
	FOLIAR APPLICATIONS: 1 to 8 quarts/acre to control broadleaf weeds and woody plants	
	BROADCAST TREATMENTS FOR FOREST SITE PREPARATION (*NOT FOR CONIFER RELEASE*): 4 to 8 quarts/acre to control susceptible woody plants and broadleaf weeds	
	CONIFER RELEASE: 4 to 20 qts in enough water to make 100 gallons of spray mixture	

Appendix 1: Triclopyr Formulations with Forestry Applications (C&P Press 2002)		
Brand Name Company/Composition	**Application Rate (Specified by Label)**	**Inerts (Specified)**
Garlon 4 Specialty Herbicide **DOW AGROSCIENCES** ***Maximum Application Rate: 8 quarts/acre*** 61.6% triclopyr (**butoxy ethyl ester**) 38.4% inert ingredients 4 lbs a.e./gallon liquid formulation **Recommends use of surfactant for best results with all spray mixtures** ***Aerial applications by helicopter ONLY***	**FOLIAR APPLICATIONS:** 1 to 8 qt/acre to control broadleaf weeds and woody plants	38.4% total other ingredients including: kerosene CAS No. 8008-20-6 proprietary surfactants contains petroleum distillates
	HIGH VOLUME FOLIAR TREATMENT WITH GROUND EQUIPMENT:: 1 to 3 qt/100 gallons of water; apply at a volume of 100 to 400 gallons of total spray/acre, depending on size and density of woody plants **LOW VOLUME FOLIAR TREATMENT WITH GROUND EQUIPMENT:** mix up to 20 qts in 10 to 100 gallons of finished spray	
	BROADCAST APPLICATION WITH GROUND EQUIPMENT:: **WOODY PLANT CONTROL**: **FOLIAGE TREATMENT**: Use 4 to 8 quarts in enough water to make 5 or more gallons/acre of total spray **BROADLEAF WEED CONTROL**: 1 to 4 quarts in a total volume of 5 or more gallons/acre as a water spray mixture	
	FOLIAGE TREATMENT (UTILITY AND PIPELINE RIGHTS OF WAY): Use 4 to 8 quarts and apply in a total spray volume of 10 to 30 gallons/acre	
	FOREST MANAGEMENT APPLICATIONS: 5 to 25 gallons/acre by air or 10 to 100 gallons/acre by ground	
	BROADCAST TREATMENTS FOR FOREST SITE PREPARATION (*NOT FOR CONIFER RELEASE*): 4 to 8 quarts/acre to control susceptible woody plants and broadleaf weeds	
	DIRECTED APPLICATIONS FOR CONIFER RELEASE: mix 4 to 20 quarts in enough water to make 100 gallons of spray mixture; spray mixture should be directed on to foliage of competitive hardwoods using knapsack or backpack sprayers with flat fan nozzles or equivalent	
	BROADCAST APPLICATIONS FOR CONIFER RELEASE IN THE EASTERN UNITED STATES: 1.5 to 3.0 quarts/acre	
	BROADCAST APPLICATIONS FOR CONIFER RELEASE IN THE LAKE STATES REGION: 1.5 to 3.0 quarts/acre	
	BROADCAST APPLICATIONS FOR CONIFER RELEASE IN THE PACIFIC NORTHWEST AND CALIFORNIA: **ON DORMANT CONIFERS BEFORE BUD SWELL (EXCLUDING PINES):** 1 to 2 quarts/acre **ON CONIFER PLANTATIONS (EXCLUDING PINES):** 1.0 to 1.5 quarts/acre	

Appendix 1: Triclopyr Formulations with Forestry Applications (C&P Press 2002)		
Brand Name Company/Composition	**Application Rate (Specified by Label)**	**Inerts (Specified)**
Garlon 3A ***Specialty Herbicide*** **DOW AGROSCIENCES** 44.4% triclopyr (3,5,6-trichloro-2-pyridinyloxyacetic acid triethylamine salt) 55.6% inert ingredients 3 lbs a.e./gallon liquid formulation **Use of an agriculturally registered non-ionic surfactant is recommended for all foliar applications; for best results, a surfactant should be added to all spray mixtures** ***Aerial applications by helicopter ONLY***	**HIGH VOLUME FOLIAR TREATMENT WITH GROUND EQUIPMENT:**: ½ to 1 gallon in water to make100 gallons of spray solution; apply at a volume of 100 to 400 gallons of total spray/acre, depending on size and density of woody plants. **LOW VOLUME FOLIAR TREATMENT WITH GROUND EQUIPMENT:** mix up to 5 gallons in 10 to 100 gallons of finished spray.	55.6% total other ingredients including: ethanol CAS No. 000064-17-8 triethylamine CAS No. 000121-44-6 ethylenediaminetetra acetic acid (EDTA) CAS No. 000060-00-4
	BROADCAST APPLICATION WITH GROUND EQUIPMENT:: **WOODY PLANT CONTROL**: **FOLIAGE TREATMENT**: Use 2 to 3 gallons in enough water to make 20 to 100 gallons/acre of total spray **BROADLEAF WEED CONTROL**: 1/3 to 1 ½ gallons in total voume of 20 to 100 gallons/acre as a water spray mixture	
	DIRECTED SPARY APPLICATIONS FOR CONIFER RELEASE: mix 1 to 5 gallons in enough water to make 100 gallson fo spray mixture	
	FOREST MANAGEMENT APPLICATIONS: recommended spray volumes are usually 10 to 25 gallons/acre by air or 10 to 100 gallons/acre by ground	
	FOREST SITE PREPARATION (*NOT FOR CONIFER RELEASE*): 2 to 3 gallons applied in a total spray volume of 10 to 30 gallons/acre	
	BROADCAST APPLICATION FOR CONIFER RELEASE IN THE NORTHEASTERN UNITED STATES:2 to 4 quarts/acre to release sprice, fir, red pine and white pine from competing hardwoods	
	BROADCAST APPLICATIONS FOR DOUGLAS FIR RELEASE IN THE PACIFIC NORTHWEST AND CALIFORNIA: 1 1/3 to 2 quarts/acre	
	CHRISTMAS TREE PLANTATIONS: 2 to 5 pints/acre as a foliar spray directed toward the base of the Christmas trees	
	DIRECTED APPLICATIONS: mix 4 to 20 fl oz in enough water to make 3 gallons of spray mixture; spray mixture should be directed on to foliage of competitie hardwoods using knapsack or backpack sprayers with flat fan nozzles or equivalent	
Pathfinder II ***Specialty Herbicide*** **DOW AGROSCIENCES** **A ready-to-use herbicide for the control of woody plants** 13.6% triclopyr (butoxyethyl ester) 86.4% inert ingredients 0.75 lbs a.e./gallon	**Forest Uses** include **Low Volume Basal Bark Treatment, Treatment of Cut Stumps, Streamline Basal Bark Treatment (Southern States)** for which applications rates expressed in units/acre are not relevant.	86.4% total, including proprietary solvent

Appendix 1: Triclopyr Formulations with Forestry Applications (C&P Press 2002)		
Brand Name Company/Composition	**Application Rate (Specified by Label)**	**Inerts (Specified)**
Remedy RTU **DOW AGROSCIENCES** **A ready-to-use brush killer: Basal Bark Applications, Sut Stump Treatments, No mixing.** 13.6% triclopyr (butoxyethyl ester) 86.4% inert ingredients 0.75 lbs a.e./gallon	**Forest Uses** include **Low Volume Basal Bark Treatment, Treatment of Cut Stumps, Streamline Basal Bark Treatment (Southern States)** for which applications rates expressed in units/acre are not relevant.	86.4% total, including proprietary solvent

Appendix 2: Forest Service Use of Triclopyr in 2001 (sorted by region) (USDA/FS 2002) [1].

Region	Forest	Use	Acres	Pounds a.e.	lb a.e./ac
	1	4 Noxious Weed Control	21	1.69	0.08
		5 Noxious Weed Control	3.3	0.52	0.16
		17 Noxious Weed Control	133	98.313	0.74
Min					0.08
Max					0.74
Total			157.3	100.52	0.64
	2	2 Noxious Weed Control	24	54	2.25
		4 Noxious Weed Control	23.68	17.35	0.73
		7 Noxious Weed Control	41.5	409.95	9.88
		14 Noxious Weed Control	4	2.25	0.56
		15 Noxious Weed Control	10.48	7.67	0.73
Min					0.56
Max					9.88
Total			103.66	491.22	4.74
	3	3 Noxious Weed Control	20	15	0.75
		6 Noxious Weed Control	100	45	0.45
		10 Noxious Weed Control	12	8	0.67
Min					0.45
Max					0.75
Total			132	68	0.52
	4	2 Noxious Weed Control	167.7	15.9	0.09
		3 Noxious Weed Control	0.25	0.5	2.00
Min					0.09
Max					2.00
Total			167.95	16.4	0.10
	5	15 Noxious Weed Control	72	17.4	0.24
		7 Noxious Weed Control	12	7	0.58
		1 Noxious Weed Control	1	2	2.00
Min					0.24
Max					2.00
Total			85	26.4	0.31

Appendix 2: Forest Service Use of Triclopyr in 2001 (sorted by region) (USDA/FS 2002) [1].

Region	Forest	Use	Acres	Pounds a.e.	lb a.e./ac
6	18	Noxious Weed Control	8.75	18.04	2.06
	21	Noxious Weed Control	150	160.5	1.07
	6	Noxious Weed Control	38	6.9	0.18
	22	Noxious Weed Control	5	4	0.80
	22	Wildlife Habitat Improvement	60	90	1.50
	17	Noxious Weed Control	8	6	0.75
	12	Noxious Weed Control	45	93	2.07
	16	Noxious Weed Control	9	1	0.11
	11	Noxious Weed Control	2.5	0.9375	0.38
	10	Noxious Weed Control	1.22	0.6699	0.55
Min					0.11
Max					2.07
Total			327.47	381.05	1.16
8	7	Conifer and Hardwood Release	2110	660	0.31
	10	Conifer Release	342	220	0.64
	9	Conifer Release	581	315	0.54
	6	Conifer Release	63	61.5	0.98
	8	Hardwood Release	182	9	0.05
	4	Hardwood control	81	60	0.74
	7	Hardwood Release	20	3.75	0.19
	4	Hardwood Release	190	100	0.53
	8	Housekeeping/Facilities Maintenance	0.2	0.7	3.50
	10	Noxious Weed Control	128	113	0.88
	8	Noxious Weed Control	1	1	1.00
	5	Noxious Weed Control	5.5	0.48	0.09
	7	Noxious Weed Control	131	444	3.39
	12	Noxious Weed Control	2	4	2.00
	7	Right-of-Way Vegetation Management	510	170	0.33
	8	Right-of-Way Vegetation Management	108.2	380	3.51
	4	Right-of-Way Vegetation Management	8.75	90	10.29
	8	Right-of-Way Vegetation Management	3.6	5.4	1.50
	7	Seed Orchard Protection	1	1	1.00
	8	Site preparation	213	80	0.38
	7	Site preparation	130	109.5	0.84
	10	Site preparation	2394	1434.6	0.60
	7	Wildlife Habitat Improvement	1173	1417.7	1.21
	12	Wildlife Habitat Improvement	891	539.5	0.61
Min					0.05
Max					10.29

Appendix 2: Forest Service Use of Triclopyr in 2001 (sorted by region) (USDA/FS 2002) [1].

Region Forest Use	Acres	Pounds a.e.	lb a.e./ac
Total	9269.25	6220.1	0.67

Appendix 2: Forest Service Use of Triclopyr in 2001 (sorted by region) (USDA/FS 2002) [1].

Region	Forest	Use	Acres	Pounds a.e.	lb a.e./ac
9	5	Right-of-Way Vegetation Management	115	367	3.19
	5	Housekeeping/Facilities Maintenance	1	1	1.00
	19	Right-of-Way Vegetation Management	0.3	0.02	0.07
Min					0.07
Max					3.19
Total			116.3	368.02	3.16
Grand Min					0.05
Grand Max					10.29
Grand Total			10358.93	7671.7	0.74

[1] Under lbs/acre, the "total" column gives the average application rate based on the total number of lbs applied divided by the total number of acres.

Appendix 3: Forest Service Use of Triclopyr in 2001 (sorted by use) (USDA/FS 2002) [1].

Use	Region	Forest	Acres	Pounds a.e.	lbs a.e. /acre
Conifer or Hardwood Release	8	4	190	100	0.53
Conifer or Hardwood Release	8	6	63	61.5	0.98
Conifer or Hardwood Release	8	7	2110	660	0.31
Conifer or Hardwood Release	8	7	20	3.75	0.19
Conifer or Hardwood Release	8	8	182	9	0.05
Conifer or Hardwood Release	8	9	581	315	0.54
Conifer or Hardwood Release	8	10	342	220	0.64
Conifer or Hardwood Release Min					0.05
Conifer or Hardwood Release Max					0.98
Conifer or Hardwood Release Total			3488.00	1369.25	0.39
Hardwood control	8	4	81.00	60.00	0.74
Hardwood control Min					0.74
Hardwood control Max					0.74
Hardwood control Total			81.00	60.00	0.74
Housekeeping/Facilities Maintenance	8	8	0.20	0.70	3.50
Housekeeping/Facilities Maintenance	9	5	1.00	1.00	1.00
Housekeeping/Facilities Maintenance Min					1.00
Housekeeping/Facilities Maintenance Max					3.50
Housekeeping/Facilities Maintenance Total			1.20	1.70	1.42
Noxious Weed Control	1	4	21.00	1.69	0.08
Noxious Weed Control	1	5	3.30	0.52	0.16
Noxious Weed Control	1	17	133.00	98.31	0.74
Noxious Weed Control	2	2	24.00	54.00	2.25
Noxious Weed Control	2	4	23.68	17.35	0.73
Noxious Weed Control	2	7	41.50	409.95	9.88
Noxious Weed Control	2	14	4.00	2.25	0.56
Noxious Weed Control	2	15	10.48	7.67	0.73
Noxious Weed Control	3	3	20.00	15.00	0.75
Noxious Weed Control	3	6	100.00	45.00	0.45
Noxious Weed Control	3	10	12.00	8.00	0.67
Noxious Weed Control	4	2	167.70	15.90	0.09
Noxious Weed Control	4	3	0.25	0.50	2.00
Noxious Weed Control	5	1	1.00	2.00	2.00
Noxious Weed Control	5	7	12.00	7.00	0.58
Noxious Weed Control	5	15	72.00	17.40	0.24
Noxious Weed Control	6	6	38.00	6.90	0.18
Noxious Weed Control	6	10	1.22	0.67	0.55
Noxious Weed Control	6	11	2.50	0.94	0.38
Noxious Weed Control	6	12	45.00	93.00	2.07
Noxious Weed Control	6	16	9.00	1.00	0.11

Appendix 3: Forest Service Use of Triclopyr in 2001 (sorted by use) (USDA/FS 2002) [1].

Use	Region	Forest	Acres	Pounds a.e.	lbs a.e. /acre
Noxious Weed Control	6	17	8.00	6.00	0.75
Noxious Weed Control	6	18	8.75	18.04	2.06
Noxious Weed Control	6	21	150.00	160.50	1.07
Noxious Weed Control	6	22	5.00	4.00	0.80
Noxious Weed Control	8	5	5.50	0.48	0.09
Noxious Weed Control	8	7	131.00	444.00	3.39
Noxious Weed Control	8	8	1.00	1.00	1.00
Noxious Weed Control	8	10	128.00	113.00	0.88
Noxious Weed Control	8	12	2.00	4.00	2.00
Noxious Weed Control Min					0.08
Noxious Weed Control Max					9.88
Noxious Weed Control Total			1180.88	1556.07	1.32
Right-of-Way Vegetation Management	8	4	8.75	90.00	10.29
Right-of-Way Vegetation Management	8	7	510.00	170.00	0.33
Right-of-Way Vegetation Management	8	8	108.20	380.00	3.51
Right-of-Way Vegetation Management	8	8	3.60	5.40	1.50
Right-of-Way Vegetation Management	9	5	115.00	367.00	3.19
Right-of-Way Vegetation Management	9	19	0.30	0.02	0.07
Right-of-Way Vegetation Management Min					0.07
Right-of-Way Vegetation Management Max					10.29
Right-of-Way Vegetation Management Total			745.85	1012.42	1.36
Seed Orchard Protection	8	7	1.00	1.00	1.00
Seed Orchard Protection Min					1.00
Seed Orchard Protection Max					1.00
Seed Orchard Protection Total			1.00	1.00	1.00
Site preparation	8	7	130.00	109.50	0.84
Site preparation	8	8	213.00	80.00	0.38
Site preparation	8	10	2394.00	1434.60	0.60
Site preparation Min					0.38
Site preparation Max					0.84
Site preparation Total			2737.00	1624.10	0.59
Wildlife Habitat Improvement	6	22	60.00	90.00	1.50
Wildlife Habitat Improvement	8	7	1173.00	1417.65	1.21
Wildlife Habitat Improvement	8	12	891.00	539.50	0.61
Wildlife Habitat Improvement Min					0.61
Wildlife Habitat Improvement Max					1.50
Wildlife Habitat Improvement Total			2124.00	2047.15	0.96
Grand Min					0.05
Grand Max					10.29
Grand Total			10358.93	7671.69	0.74

Appendix 3: Forest Service Use of Triclopyr in 2001 (sorted by use) (USDA/FS 2002) [1].

Use	Region	Forest	Acres	Pounds a.e.	lbs a.e. /acre

[1] Under lbs/acre, the "total" column gives the average application rate based on the total number of lbs applied divided by the total number of acres.

Appendix 4: Acute toxicity of triclopyr and triclopyr formulations to experimental mammals.

Species	Exposure	Response	Reference
		ORAL	
Triclopyr, technical			
Rats, Sprague Dawley, males and females, fasted wgt on day of dosing = 203-298 g (males) and 163-204g (females)	Single oral dose of 1200 (males only), 2000, or 5050 mg/kg technical grade triclopyr	No mortality at 1200 or 2000 mg/kg. Clinical signs of toxicity included decreased activity, diarrhea, hunched posture, polyuria, facial swelling, stained fur, and walking on tiptoe, which were no longer evident in survivors at day 6. Abnormal necropsy findings in animals that died pertained to fur, testes, lungs, liver, and contents of the GI tract. In animals that were sacrificed, abnormal necropsy findings pertained to lungs, liver, hearts, kidneys, and contents of the GI tract. LD_{50} = 1915 mg/kg (males) LD_{50}>2000 but <5050 mg/kg (females)	Kuhn 2001 MRID 45451304
Triclopyr, free acid form			
Rats, male	729 mg/kg	LD_{50} =729 mg/kg	MRID 00031940
Rats, female	630 mg/kg	LD_{50} =630 mg/kg	MRID 00031940
Triclopyr, TEA			
Rats, Fischer, male and female	1000, 2500 or 3200 mg/kg (Garlon 3A, 32.2% w/w triclopyr)	LD_{50} =2574 mg/kg or 828 mg a.e./kg (males) LD_{50} =1847 mg/kg or 594 mg a.e./kg (females)	Mizell and Lomax 1988 MRID 41443301

Appendix 4: Acute toxicity of triclopyr and triclopyr formulations to experimental mammals.

Species	Exposure	Response	Reference
Triclopyr, BEE			
Rats, male and female	803 mg/kg (97.1% a.i.)	LD_{50} =803 mg/kg	MRID 00031940
Triclopyr, NOS			
Horse Adult Shetland pony geldings, 151-203 kg 3 in control and 6 in each dosed group.	Acid administered by gavage in corn oil:acetone vehicle. Vehicle controls used. Daily doses of 0, 60, and 300 mg/kg for 4 days. Six day post-treatment observation period.	No clinical signs of toxicity at 60 mg/kg [Cumulative dose of 240 mg/kg]. At 300 mg/kg [Cumulative dose of 1200 mg/kg], signs of toxicity included depression and recumbency. Decrease GI activity. Increased and labored respiration with cyanotic mucus membranes in some animals. Ataxia, stiffness and weakness with fine tremors. Slight changes in blood urea nitrogen, blood glucose, serum calcium, and serum iron. Pale liver and swollen kidneys. Mild to moderate hepatosis and cellular swelling and fatty changes around the central veins of the liver. Vacuolar swelling and cast formation in the renal tubules at 300 mg/kg. At 300 mg/kg, 2/6 ponies died on days 5 and 6 of study and a third pony was euthanized on day 5. Another pony, moderately affected, was euthanized on day 6. The remaining 2 ponies were only mildly affected. Estimated LD_{50} = >1000 mg/kg.	Osweiler 1983

Appendix 4: Acute toxicity of triclopyr and triclopyr formulations to experimental mammals.

Species	Exposure	Response	Reference
		DERMAL	
Triclopyr, technical			
Rabbits, New Zealand White, 5 males and 5 females, weighing 2.200-2.600 kg (males) and 2.550-3.275 kg (females)	Dermal application of 5050 mg/kg technical grade triclopyr to intact skin	No mortality; no clinical signs of toxicity. The only sign of dermal irritation was erythema in 4/10 animals. No effects on body weight gain. Abnormal necropsy findings in 5/10 animals pertained to lungs and kidneys. LD_{50}>5050 mg/kg (Toxicity Category IV)	Kuhn 2000a MRID 45451305
Rabbits, New Zealand White,3 males and 3 females, weighing 2.400-2.550 kg (males) and 2.275-2.750 kg (females)	Dermal application of 500 mg of triclopyr technical to intact skin, which was covered with semi-permeable dressing, and maintained in contact with the skin for 4 hours	slightly irritating (Toxicity Category IV)	Kuhn 2000c MRID 45451308
Guinea pigs, Hartley-albino, 10 males and 10 females, weighing 360-411 g (males) or 342-393 g (females)	Challenged with dermal dose of 400 mg triclopyr technical after treatment 1/week for 3 weeks with 400 mg triclopyr technical	No irritation	Kuhn 2000d MRID 45451309
Triclopyr, free acid form			
Rabbits	>2000 mg/kg	LD_{50} >2000 mg/kg	MRID 00056009
Triclopyr, TEA			
Rabbits, New Zealand white, males and females	>2000 mg/kg (Garlon 3A)	LD_{50} >2000 mg/kg No effects on weight gain or gross pathology. On male dies from an undetermined cause.	Mizell and Lomax 1989 MRID 41443302
Rabbits, New Zealand white, six	primary dermal irritation study with Garlon 3A	Not irritating	Mizell 1988b MRID 41443305

Appendix 4: Acute toxicity of triclopyr and triclopyr formulations to experimental mammals.

Species	Exposure	Response	Reference
Rabbits, New Zealand white, six/sex	Garlon 3A (46.5% TEA as a.i.) dermal exposure for 24 hours	LD_{50} >5000 mg/kg No mortality or changes in gross pathology.	Gilbert 1996 MRID 43952401
Guinea pigs, Hartley, males, weighing 300-360 g	Garlon 3A in dermal sensitization study	10 of 10 positive controls challenged with DER 331 had slight to moderate redness Under identical conditions, none of the 10 guinea pigs challenged with the undiluted test material showed any signs of redness or edema. Study concludes that Garlon 3A is not considered a potential human skin sensitizer	Carreon 1985 MRID 40055701
Guinea pigs, Hartley, males, 10	dermal sensitization study using 50% Garlon 3A	sensitizer Challenge application of 50% Garlon 3A caused slight erythema in 4 of 10 animals	Mizell 1989 MRID 41443306
Guinea pigs, Hartley, males, 10	dermal sensitization study using 30% Garlon 3A	not sensitizing 30% Garlon 3A did not cause delayed contact hypersensitivity in guinea pigs	Berdasco 1990b MRID 41830602
Guinea pigs, Hartley, males, 10	dermal sensitization study using 15% Garlon 3A	not sensitizing 15% Garlon 3A did not cause delayed contact hypersensitivity in guinea pigs	Berdasco 1990a MRID 41830601

Appendix 4: Acute toxicity of triclopyr and triclopyr formulations to experimental mammals.

Species	Exposure	Response	Reference
Guinea pigs, Hartley, 10/sample	dermal sensitization potential with four samples of 0. 4 mL of Garlon 3A, each containing a different level of a contaminant known as ethyl pyridone (in ascending order) as the challenge dose. Positive controls received a challenge dose of 7.5% DER 331	Challenge application with the positive control caused slight erythema at the test site in 10/10 animals. Challenge application with 0.4 mL of Garlon 3A caused slight erythema in 3/10, 4/10, 1/10, and 2/10 animals. Investigators conclude that Garlon 3A has the potential to cause delayed contact hypersensitivity in guinea pigs, but that this potential is not associated in a dose-related manner with the level of ethyl pyridone in the sample.	Berdasco 1994a MRID 43230202
Triclopyr, BEE			
Guinea pigs, Hartley, 10/sample	dermal sensitization potential with four samples of 0. 4 mL of 10% or 5% Garlon 4, each containing a different level of a contaminant known as ethyl pyridone (in ascending order) as the challenge dose. Positive controls received a challenge dose of 7.5% DER 331	Challenge application with the positive control caused slight erythema at the test site in 10/10 animals. Challenge application with 0.4 mL of 10% of 5% Garlon 4 caused slight erythema in 4/10, 3/10, 3/10, and 1/10 animals. Investigators conclude that Garlon 4 has the potential to cause delayed contact hypersensitivity in guinea pigs, but that this potential is not associated in a dose-related manner with the level of ethyl pyridone in the sample.	Berdasco 1994b MRID 43230203
Guinea pigs, Hartley, males, 10	dermal sensitization study using 2.5% Garlon 4	not sensitizing 2.5% Garlon 4 did not cause delayed contact hypersensitivity in guinea pigs	Berdasco 1990c MRID 41830603

Appendix 4: Acute toxicity of triclopyr and triclopyr formulations to experimental mammals.

Species	Exposure	Response	Reference
Guinea pigs, Hartley, males, 10	dermal sensitization study using 7.5% Garlon 4	not sensitizing 7.5% Garlon 4 did not cause delayed contact hypersensitivity in guinea pigs	Berdasco 1990a MRID 41830601
Rabbits	>2000 mg/kg	LD_{50} >2000 mg/kg	MRID 40557005
Rabbits, white	primary dermal irritation study	Not irritating	MRID 40557008
Rats, Fischer 344, 5 males and 5 females, initial weight of 120-180 g	Dermal exposure to5000 mg/kg bw of NAF-5 (Pathfinder II) (13.9% a.i.) to shaved backs of rats for 24 hours.	LD_{50} >5000 mg/kg bw No mortality; no treatment-related gross pathology	Brooks and DeWildt 2000 MRID 45181402
Guinea pigs	dermal sensitization study	sensitizer	MRID 40557009

INHALATION

Triclopyr, technical

Rat, Sprague-Dawley, 5 males and 5 females, weighing 247-324 g (males) or 186-220 g (females)	4-hour exposure to undiluted test substance (triclopyr technical) (fine powder) at 2.56 mg/L	No mortality. Clinical signs of toxicity included decreased activity and piloerection, no longer evident by day 1. No effects on body weight except in one male during the week 1. No abnormal necropsy findings except spotted lungs in one rat. LC_{50} >2.56 mg/L (Toxicity Category IV)	Carter 2000 MRID 45451306

Triclopyr, TEA

Rats, male and female	>2.6 mg/L	LC_{50} >2.6 mg/L	MRID 41443303

Triclopyr, BEE

Rats, male and female	>4.8 mg/L	LC_{50} >4.8 mg/L	MRID 40557006

Appendix 4: Acute toxicity of triclopyr and triclopyr formulations to experimental mammals.

Species	Exposure	Response	Reference
OCULAR			
Triclopyr Technical			
Rabbits, New Zealand White, 3 males and 3 females, weighing 2.000-2.350 kg (males) or 2.250-2.300 kg (females)	Application of 0.1 mL by volume (42.7 mg) to conjunctival sac of right eye for 24 hours, followed by 1 minute of washing with room temperature deionized water	mild irritation (Toxicity Category II) No positive effects at 7 days after treatment.	Kuhn 2000b MRID 45451307
Triclopyr TEA			
Rabbits, New Zealand white, six	primary eye irritation study	corrosive	Mizell 1988a MRID 41443304
Triclopyr, BEE			
Rabbits	primary eye irritation study	minimally irritating	MRID 40557007
INTRAVENOUS			
Triclopyr Butotyl (ACTP) Ester			
Goats, black Bengal, 1.5- to 2-years old, weighing 9.5-12 kg, one male and one female per dose group	0, 2.97, 5.94, or 11.88 mg/kg corresponding to 1/240, 1/120, or 1/60 of the LD_{50} of triclopyr acid (713 mg/kg)	No signs of toxicity at 2.97 or 5.94 mg/kg. At 11.88 mg/kg, signs of toxicity included depression and drowsiness after 10 minutes, miosis and fixation of the eyelid, increased secretion of nasal discharge and salivation, irregular skin itching, yarning, muscle tremors mainly on the posterior portion of the body, slight increase of body temperature, and increased frequency of defecation until 4 ½ hours after administration of test substance. 11.88 mg/kg ACTP = minimum intravenous toxic dose.	Sar et al. 2002

Appendix 5: Systemic toxicity of triclopyr after repeated oral administrations.

Species	Exposure/Response	Reference
Rats, Fischer 344, males and females	Exposure: Dietary concentrations of triclopyr technical (98% a.i.) at doses of 0, 5, 20, 50, or 250 mg/kg/day for 13 weeks. Response: At equal to or greater than 20 mg/kg/day, increased incidence of proximal tubule degeneration of the kidneys in both sexes; at 50 mg/kg/day, significant increase in absolute and relative kidney weight in males; at 250 mg/kg/day, increase in relative kidney weight in males and females. Slight decrease in body weight in females at 250 mg/kg and in males at 50 and 250 mg/kg. Dose/severity related degeneration of the proximal tubules of the kidneys at dose equal to or greater than 20 mg/kg. This was accompanied by an increase in kidney weight. Slight functional changes in kidneys at 250 mg/kg. Centrilobular liver cells of male rats at 250 mg/kg were slightly more eosinophilic than controls. This was accompanied by a slight elevation of SGPT and a decrease in serum proteins. Systemic NOEL = 5 mg/kg/day Systemic LOEL = 20 mg/kd/day, based on histopathological changes in kidneys of male and female rats.	Landry et al. 1984 MRID 00150378

Appendix 5: Systemic toxicity of triclopyr after repeated oral administrations.

Species	Exposure/Response	Reference
Rats, Fischer 344, weanling, male and female, 10/sex/dose group	Exposure: Triclopyr BEE in the diet at dose levels of 0, 7, 28, 70, or 350 mg/kg bw/day for 13 weeks. Response: 70 mg/kg bw/day caused histopathological renal alterations and elevated relative kidney weights in males, but not females. 350 mg/kg bw/day caused lower body weights and feed consumption, elevated relative kidney weights, and degeneration/regeneration fo the descending portion of the renal proximal tubules in both males and females. Furthermore, hepatic toxicity in both males and females at 350 mg/kg bw/day was evidenced by minor elevations in ALP, ALT, and AST values, elevated relative liver weights,a nd histopathological changes. NOEL = 28 mg/kg bw/day triclopyr BEE in males for subchronic dietary exposure NOEL = 70 mg/kg bw/day triclopry BEE in females for subchronic dietary exposure The investigators indicate that the results of this study are consistent with the results reported by Landry et al. (1984) which uses the same strain of rats and equivalent dose levels of triclopyr (acid).	Barna-Lloyd et al. 1992 MRID 42274901

Appendix 5: Systemic toxicity of triclopyr after repeated oral administrations.

Species	Exposure/Response	Reference
Rats, Fischer 344, male and female, 50/sex/dose group	Exposure: Dietary concentrations of triclopyr technical (98.0% a.i.) at dose levels of 0, 3, 12, or 36 mg/kg/day for 2 years.[Additional groups of 10 rats/sex/dose group received dietary exposure to same dose levels of triclopyr for 6 and 12 months.]	Eisen-brandt et al. 1987 MRID 40107701

Additional notes on Eisen-brandt et al. 1987 MRID 40107701:

Response: No treatment-related mortality; statistically significant decreases observed in red cells at 12 months, in hemoglobin at 6 months, and in hematocrit at 6 and 12 months. Absolute and relative kidney weights significantly increased (10-17%) in high dose (36 mg/kg/day) male rats, with an apparent dose-related trend at 12 months. Female rats at all dose levels had an increased incidence of pigmentation of the proximal descending tubule, compared with controls, while male rats in the 6-month treatment group had an increased incidence of proximal tubule degeneration at the 12 and 26 mg/kg/day dose levels, compared with controls.

NOEL for chronic toxicity = 12 mg/kg/day for males and 36 mg/kg/day for females.

LOEL = 36 mg/kg/day for males based on marginal increases in proximal tubular degeneration at 6 months.

No significant trends in tumor incidence for male rats. There were significant pair-wise differences vs controls at 3 and 12 mg/kg triclopyr in the incidence of adrenal gland benign pheochromocytomas and benign and/or malignant pheochromo-cytomas combined and in the incidence of skin fibromas at 3 and 12 mg/kg/day ($p<0.05$) for all comparisons except the incidence of pheochromocytoma (benign and combined) at 12 mg/kg ($p<0.01$ vs controls).

Female rats had significant increasing trends in mammary gland adenocarcinomas ($p<0.05$) and in adenomas and/or adnenocar-cinomas combined ($p<0.01$). There was a significant difference in the pair-wise comparison fo the 36 mg/kg/day dose group with controls for mammary gland adenomas and/or adenocarcinomas combined ($p<0.05$). There were no significant pair-wise comparisons or trends for the incidence of adrenal gland pheochromocytomas in female rats.

Appendix 5: Systemic toxicity of triclopyr after repeated oral administrations.

Species	Exposure/Response	Reference
Rats, F344	Exposure: Administered in the diet at concentrations which resulted in doses of 0, 7, 28, 70, and 350 mg/kg bw/day as butoxyethyl ester for 90 days. 9 animals/sex/dose. Response: Decreased body weight and hematologic changes in males in high dose group. Increased relative kidney and liver weight in males at 28 and 70 mg/kg. Hematologic changes as well as increases in relative liver and kidney weight in females at 7 mg/kg/day. Histopathological changes in the liver and kidney of males at 70 and 350 mg/kg and in females at 350 mg/kg. Systemic LEL of 28 mg/kg for males and ≤ 7 mg/kg for females.	Barna-Lloyd et al. 1992 (MRID No. 42274901)
Dogs, beagle, male and female, 4 dogs/sex/dose group	Exposure: Dietary doses of triclopyr technical (a.i. not specified) at 0, 0.1, 1, 0.5, or 2.5 mg/kg/day for 183 days (males) or 184 days (females). Response: No significant treatment related effects on body weight, food consumption, hematology, or clinical chemistry in male or females; at 2.5 mg/kg/day, a decreased rate of phenolsulfonthalein (PSP) excretion was observed; however, the effect, later determined to be the result of triclopyr and PSP competing for renal excretion, was not considered toxicologically relevant. Systemic NOEL equal to or greater than 2.5 mg/kg/day Systemic LOEL equal to or greater than 2.5 mg/kg/day in both sexes	Quast et al. 1977 MRID 00071794

Appendix 5: Systemic toxicity of triclopyr after repeated oral administrations.

Species	Exposure/Response	Reference
Dogs, beagle, male and female, 14 months old, 4 dogs/sex/dose group	Exposure: Dietary concentrations of DOWCO 233 [triclopyr technical] at doses of 0, 5, 10, or 20 mg/kg/day for 228 days. Response: NOEL = 10 mg/kg/day LOEL = 20 mg/kg/day for male and female dogs, based on decreased body weight gain and decreased hematological parameters in males; changes in clinical chemistry in males and females, and liver histopathology in male and female dogs. Decreased body weight and food consumption in females at all dose levels; slight thinning of coat hair in females at 10 and 20 mg/kg/day; decrease in erythroid values in males and females at 20 mg/kg/day; effects on clinical chemistry, including increased SGPT activity in all females at all dose levels and in males at 20 mg/kg/day, increased AP and OCT activity in females at 20 mg/kg/day, increased SGOT activity in males and females at 20 mg/kg/day, and decreased renal excretion of PSP dye in males and females at all dose levels. [A 38-day supplemental study suggested the existence of a competitive mechanism of renal excretion for the test material and the PSP dye at dose levels of 1 or 2, but not 0.5, mg/kg/day.] Principal organ weight changes included increased relative liver weights in males at 10 and 20 mg/kg/day and in females at 20 mg/kg/day; relative kidney weight were increased in females at 10 and 20 mg/kg/day; necropsy examination revealed decreased amounts of adipose tissue in females at 20 mg/kg/day; microscopic examinations revealed minimal (reversible) degenerative changes in liver and kidneys in males and females at all dose levels.	Quast et al. 1976 MRID 00071793
Dogs, beagle, male and female, 14 months old, 4 dogs/sex/dose group	This is a supplemental study reported with the previous study. The supplemental study was designed to evaluated the effects of DOWCO 233 on PSP excretion in dogs. The NOAEL for an inhibition of secretion of PSP (given either in gelatin capsules or by incorporation into the diet) by male dogs was 0.5 mg/kg/day. A dose of 2 mg/kg/day resulted in a slight inhibition of PSP secretion. Inhibition of PSP secretion was reversible after a minimum of 10 days.	Quast et al. 1976 MRID 00071793

Appendix 5: Systemic toxicity of triclopyr after repeated oral administrations.

Species	Exposure/Response	Reference
Dogs, beagles, male and female, 3 months old, 4/sex/dose group	Exposure: Dietary concentrations of triclopyr technical (98.9% a.i.) at doses of 0, 0.5, 2.5, or 5.0 mg/kg/day for 1 year. Response: No significant effects of treatment on mortality, clinical signs, body weight, or food consumption in males or females at any dose levels. Statistically significant increases in creatinine (30 and 40% in males dogs at 2.5 and 5.0 mg/kg/day dose levels; 55 and 44% in females at 12 months). No histopathologic changes in the kidney. A decrease in PSP excretion in the middle- and high-dose dogs Significant increases in serum urea nitrogen and creatinine at 2.5 mg/kg. There effects were more pronounced at 5 mg/kg. No effects at 0.5 mg/kg. **This is the basis for the OPP/RfD.** Measurements of BUN PRE-TREATMENT AFTER TREATMENT Dose Males Females Males Females 0.0 16 14 16 17 0.5 13 15 18 19 2.5 14 13 22 20 5.0 13 15 27 23 **This study was used as the basis for the previous U.S. EPA/OPP (1995b) RfD on triclopyr.**	MRID 41200301 Quast et al. 1988 MRID 41200301
Mice (NOS), male and female	Exposure: Dietary concentrations of technical grade triclopyr at doses levels of 0, 200, 400, 800, 1600, or 3200 ppm (nominal doses of 30, 60, 120, 240 or 480 mg/kg/day) for 28 days (range finding study). Response: At equal to or greater than 120 mg/kg/day, dose-related adverse effects included centrilobular swelling and degeneration of hepatocytes in males and mild increases in liver enzymes at 240 mg/kg/day; at 480 mg/kg/day, effects in males included single cell necrosis of the liver, significant increases in alkaline phosphatase, AST, and ALT, and liver enlargement and dark color.	Tsuda et al. 1987 MRID 40356601

Appendix 5: Systemic toxicity of triclopyr after repeated oral administrations.

Species	Exposure/Response	Reference
Mice, ICR, 60 mice/sex/ dose group	Exposure: Dietary concentrations of triclopyr technical (98.0% a.i.) at dose levels of 0, 50, 250, or 1250 ppm corresponding to ***equivalent oral doses for males of 5.55, 28.6, or 143 mg/kg/day*** and ***equivalent oral doses for females of 5.09, 26.5 or 135 mg/kg/day*** for 95 weeks Response: In high dose males (143 mg/kg/day), water consumption increased an average of 25 % beginning at week 13; plasma BUN increased 25%, compared with controls, at 26 weeks, and liver weight increased by 17% at week 26 only. In high dose females (135 mg/kg/day), kidney weight increased 10-16%, while urinary protein was also increased at week 52. There were, however, no pathology data to support a true toxic effect on the kidney of treated males or females. A significant increasing trend in mammary gland adenocarcinomas ($p<0.05$) was observed in female mice; no compound-related tumors were observed in male mice. There were no significant differences in the pair-wise comparisons of treated groups and controls.	Tsuda et al. 1987 MRID 40356601

Appendix 6: Developmental and Reproductive Effects [1].

Species	Exposure/Response	Reference
	Multi-generation Reproduction Studies	
Rats, Sprague-Dawley, male and female, 30/sex/dose	Exposure: Two-generation reproductive study with acid form of triclopyr. Triclopyr technical (99.4%) in the diet at nominal doses of 0, 5, 25, or 250 mg/kg/day (P_1 high dose males received 100 mg/kg/day for the first 29 days of the study) for 10 weeks prior to breeding. After 10 weeks, P_1 rats were mated to produce F_1 litters. After weaning, groups of 30 male and 30 female F_1 pups were randomly selected to become the second parental (P_2) generation. After approximately 12 weeks of dietary exposure the P_2 adults were mated to produce the F_2 litters.	Vedula et al. 1995 MRID 43545701

Additional Notes on Vedula et al. 1995

Response: No adverse treatment-related effects observed on any parameter in adult and neonatal males or females given 5 mg triclopyr/kg/day; no reproductive or developmental effects noted at 25 mg/kg/day; decrease in adult male and female feed consumption and body weights throughout the study at 250 mg/kg/day, compared with controls.

Treatment related increases in relative kidney weights observed at 25 (P_1 males only) or 250 mg/kg/day (P_1 and P_2 male and females); decreased liver weights in adult rats at 250 mg/kg/day; kidneys identified pathologically as the target organ for toxicity in both generations of adult rats; treatment related degeneration of renal proximal tubules in some male and female rats at 25 mg/kg/day and the majority of male and female rats at 250 mg/kg/day; no histopathological findings accompanied the decreased liver weights at 250 mg/kg/day; no treatment related gross or histopathological changes in either adult generation of male or female rats at 5 mg/kg/day; and no primary treatment related toxicological or histopathological changes on the reproductive organs in either generation of male or female rats in any dose group.

Pup weights, pup survival, and litter sizes significantly decreased at the 250 mg/kg/day dose in both generations; and fertility and conception rates in second generation males and females decreased at 250 mg/kg/day. The lower fertility rates were attributed to the female in light of the effects on litter size and pup survival and lack of effects on spermatogenesis as indicated by histopathological evaluation of the testes.

NOEL = 5 mg/kg/day for parental systemic toxicity.
NOEL = 25 mg/kg/day for fertility and neonatal toxicity

This study is the basis for the U.S. EPA/OPP (1998a) RfD for triclopyr cited in the RED.

Appendix 6: Developmental and Reproductive Effects [1].

Species	Exposure/Response	Reference
Rats, Sprague-Dawley, 35- to 40-days-old, 11-2 males per dose and 23 females per dose	Exposure: DOWCO 233 (technical grade triclopyr) at dietary concentrations adjusted to provide daily intake of 3, 10, or 30 mg test material per kg of body weight for 3 generations. Response: DOWCO 233 produced no effect on the reproductive capacity, growth, or maturation of rats. Third generation pups from one litter at 3 mg/kg/day appeared weak and evidenced retarded growth. This was associated with non-functioning mammary glands in the dam. No similar effects were observed at higher doses. [Also included in Hanley et al. 1983, MRID 00137618, and as a publication by Hanley et al. 1984.]	Beliles and Wosu 1976 MRID 00057084

Appendix 6: Developmental and Reproductive Effects [1].

Species	Exposure/Response	Reference
	Teratology Studies	
Rats, Sprague-Dawley, mated females with initial mean body weights of 205-215 g, 25 rats/dose group	Exposure: Dowco 233 [(3,5,6-trichloro-2-pyridyloxy)acetic acid] administered by gavage at doses of 0, 50, 100, or 200 mg/kg/day once daily, on days 6-15 of gestation. Response: Signs of maternal toxicity, including rough hair, salivation, occasional dyspnea and tremors, and apparent abdominal discomfort immediately after treatment were observed at all dose levels. Food consumption was decreased at 100 and 200 mg/kg/day and body weight gain was significantly depressed at 200 mg/kg/day. Treatment did not significantly affect the numbers of implantations, viable fetuses, resorptions, or corpora lutea, fetal body weights or sex ratios. Among litters from dams exposed to 200 mg/kg/day, there was a statistically significant increase in the incidence of retarded ossification of the skull bones. Also, two fetuses from the same group had major malformations, which, because of the low incidence, could not be equivocally attributed to treatment. Doses of 50 or 100 mg/kg/day, although mildly toxic to the dams, did appear to cause adverse effects in the developing fetuses. [Note: The two MRID submissions appear to cover the same study. The cover page of MRID 41688301 indicates that the "***Reformat***" is prepared by Breslin.]	Thompson et al. 1979 MRID 00072441 Breslin 1990a MRID 41688301

Appendix 6: Developmental and Reproductive Effects [1].

Species	Exposure/Response	Reference
Rats, Sprague-Dawley, female adults	Exposure: Triclopyr (3,5,6-tricloror-2-pyridyloxyacetic acid) (98.5% purity) at gavage doses of 0, 50, 100, or 200 mg/kg/day on days 6 through 15 of gestation. Response: Dose-related, transient maternal toxicity, including roughening of the hair and excessive shedding was observed in all dose groups. Body weight gain was decreased 13% in the 100 mg/kg/day group and 17% in the 200 mg/kg/day group. Food consumption was also decreased in the 100 and 200 mg/kg/day groups. No significant adverse effects were observed with respect to the number of corpora lutea, implantations, or litter size. An increased resorption rate at 200 mg/kg/day was attributable to complete resorption of one entire litter. A slight, but not statistically significant decrease in fetal body weight was observed at 200 mg/kg/day. Two fetuses from the 200 mg/kg/day dose group had major malformations – i.e., cleft palate, brachycephaly (short broad head), and various skeletal abnormalities. Minor soft tissue and skeletal variations were observed in fetuses from the control and treated groups.	Hanley et al. 1983 MRID 00137618

Appendix 6: Developmental and Reproductive Effects [1].

Species	Exposure/Response	Reference
Rats, time-mated Crl: CD(SD) BR VAF/Plus females	Exposure: Triclopyr triethylamine (TEA) salt technical (46.5% a.i.) administered at doses of 0, 30, 100, or 300 mg/kg, corrected for compound purity, on days 6 through 15 of gestation. Response: Maternal toxicity at 300 mg/kg included increased incidence of clinical signs (salivation) and mortality (1 death). Maternal NOEL = 100 mg/kg Maternal LOEL = 300 mg/kg, based on increased incidence of salivation and mortality. Developmental toxicity at 300 mg/kg was manifested as decreased mean fetal body weight, increased fetal and litter incidence of skeletal anomalies (reduced ossification), and an increased incidence in the number of fetuses with unossified sternebrae. Developmental NOEL = 100 mg/kg Developmental LOEL = 300 mg/kg, based on decreased mean fetal weight, increased fetal and litter incidence of skeletal anomalies, and increased fetal incidence of unossified sternebrae.	MRID 43217602 Bryson 1994b
Rats, Sprague-Dawley, mated females, weighing 217-221 g, 25/dose group	Exposure: Dowco 233 (triclopyr) administered by gavage at doses of 0, 50, 100, or 200 mg/kg/day once daily, on days 6-15 of gestation. Response: Signs of maternal toxicity observed in some rats from all dose groups included rough hair, salivation, occasional dyspnea. Retarded ossification at 200 mg/kg/day.	Breslin 1990a MRID 41688301

Appendix 6: Developmental and Reproductive Effects [1].

Species	Exposure/Response	Reference
Rats, CD, time-mated females	Exposure: Gavage doses of 0, 30, 100, or 300 mg/kg/day triclopyr TEA on days 6 through 15 of gestation. Response: Marked maternal toxicity at 300 mg/kg/day manifested as mortality, clinical signs, body weight loss or decreased body weight gain, increased water and decreased feed consumption, and increased kidney weights. At lower doses, signs of maternal toxicity included decreased feed consumption and increased water consumption at 100 mg/kg/day. Developmental effects included decreased fetal weight and decreased ossification at 300 mg/kg/day. No teratogenic effects. Maternal NOEL = 30 mg/kg/day Developmental NOEL = 100 mg/kg/day [Abstract [1]]	Breslin et al. 1996

Appendix 6: Developmental and Reproductive Effects [1].

Species	Exposure/Response	Reference
Rats, CD, time-mated females, 25/dose group in Phase I and 30/dose group in Phase II. **Note: Phase I and II are two separate studies. Phase II was conducted to assess the reproducib-ility of effects seen in Phase I.**	Exposure: **Phase I**: Gavage doses of 0, 30, 100, or 300 mg/kg/day triclopyr BEE (97.0% pure) on days 6 through 15 of gestation. **Phase II**: Gavage doses of 0, 5, 30, 100, or 300 mg/kg/day triclopyr BEE (97.0% pure) on days 6 through 15 of gestation. Response: At 300 mg/kg/day, signs of marked maternal toxicity including four deaths, overt clinical signs in a few dams, mean body weight loss and decreased mean body weight gain, decreased mean feed consumption, increased mean water consumption, and increased mean liver and kidney weights. At 100 and 30 mg/kg/day, slight initial reduction in body weight gain that persisted throughout the study; increased water consumption at 100 mg/kg/day. The only litter effects seen in both Phase I and Phase II was an increased incidence of extra ribs at 300 mg/kg/day. In Phase I, an increase in late in utero deaths at 300 mg/kg/day. Decreased uterine weight and litter weight at 100 and 30 mg/kg/day in Phase I only. Effect was not dose dependent. In Phase I, a dose-related increase in number of liters with malformed fetuses: 2/25, 1/23, 3/24, and 6/16 at 0, 30, 100, or 300 mg/kg/day. Abnormalities included microphthalmia/anophthamia (small or missing eyes) and other craniofacial abnormalities. Malformed litters were from dams with the most severe signs of toxicity at 300 mg/kg/day. The malformations were not seen in Phase II. NOEL for embryo-toxicity = 100 mg/kg/day **This study is cited but not discussed in U.S. EPA/OPP (1998a) RED for triclopyr. It appears to be used by U.S. EPA/OPP (2002) as the basis for the acute RfD of 1 mg/kg/day.**	Jones 1995 MRID 43675801

Appendix 6: Developmental and Reproductive Effects [1].

Species	Exposure/Response	Reference
Rabbits, New Zealand white, females	Exposure: DOWCO 233 at dose levels of 25, 50, or 100 mg/kg/day by gavage during days 6-18 of gestation, Response: Maternal toxicity, including mortality was observed at all dose levels; no evidence of toxicity to the developing embryo and fetus as a result of maternal treatment; no gross anomalies observed at any dose level; no soft tissue anomalies among litters of treated animals, compared with controls. Investigators conclude that DOWCO 233 administered to pregnant rabbits at dose levels that caused some maternal deaths was neither embryotoxic nor fetotoxic.	Smith et al. 1960 MRID 00057083
Rabbits, New Zealand white, sexually mature females	Exposure: Triclopyr (3,5,6-tricloro-2-pyridyloxyacetic acid) (98.5% purity) by gavage at dose levels of 0, 10, or 25 mg/kg/day on days 6 through 18 of gestation. Response: Transient, dose-related decreases in maternal body weight gain. No signs of treatment-related effects on fetal growth or development.	Hanley et al. 1983 MRID 00137618

Appendix 6: Developmental and Reproductive Effects [1].

Species	Exposure/Response	Reference
Rabbits, New Zealand white, females	Exposure: Triclopyr BEE technical (96.9% a.i.) administered at doses of 0, 10, 30, or 100 mg/kg/day on gestations days 6-18 inclusive. Response: Evidence of maternal toxicity at 100 mg/kg included mortality during test article administration, decreased number of live fetuses, decreased number of live fetuses/dam, increased post-implantation loss ($p<0.05$), and increased number of fetal deaths. NOEL for maternal toxicity = 30 mg/kg Maternal LEL = 100 mg/kg, based on increased mortality at this dose. Evidence of developmental toxicity at 100 mg/kg included decreased number of live fetuses, increased number of fetal deaths, and increased number of fetal and/or litter incidence of skeletal anomalies and variants. Developmental NOEL = 30 mg/kg Developmental LOEL = 100 mg/kg	MRID 43217601 Bryson 1994a
Rabbits, New Zealand, white, females	Exposure: Triclopyr BEE via gavage on days 6 through 18 of gestation at dose levels of 0, 10, 30, or 100 mg/kg/day. Response: Severe maternal toxicity at 100 mg/kg, including mortality, body weight loss, and decreased feed consumption. Developmental effects were observed only at 100 mg/kg and included increased resorption, decreased litter size and litter weight, and increases in minor skeletal alterations, specified as additional sterbral centers, reduced ossification of digital bones, and extra (13) ribs. No teratogenic effects even at maternally toxic dose levels. Maternal NOEL = 30 mg/kg/day Developmental NOEL = 30 mg/kg/day [Abstract [1]]	Breslin and Billington 1995

Appendix 6: Developmental and Reproductive Effects [1].

Species	Exposure/Response	Reference
Rabbits, New Zealand white females	Exposure: Triclopyr TEA technical (46.5% a.i.) administered at doses of 0, 10, 30, or 100 mg/kg on days 6 through 18 of gestation, inclusive. Doses were corrected for compound purity. Response: Maternal toxicity at 100 mg/kg manifested as increased mortality during test article administration, decreased body weight gain and food efficiency, and increased liver and kidney weights. Maternal NOEL = 30 mg/kg Maternal LOEL = 100 mg/kg day, based on decreased body weight gain, decreased food efficiency, and increased liver and kidney weight. Developmental toxicity at 100 mg/kg was manifested as decreased number of litters, decreased number of corpus lutea, decreased number of total implants, decreased number of total live fetuses, increased embryonic deaths and deaths/dam, and increased pre-implantation loss. Developmental NOEL = 30 mg/kg Developmental LOEL = 100 mg/kg, based on decreased number of live implants, decreased number of live fetuses, and increased embryonic deaths.	MRID 43217603 Bryson 1994c
Rabbits, New Zealand white, females	Exposure: Triclopyr TEA single bolus dose in corn oil suspension at dose levels of 0, 10, 25, or 75 mg/kg/day on days 6-18 of gestation. Response: Slight increase in maternal mortality at 75 mg/kg/day; no effects on other maternal parameters at any dose level. No treatment-related effects on observed on any developmental parameters at any dose level. NOEL = 75 mg/kg/day for developmental toxicity [Abstract [1]]	Kirk et al. 1989

Appendix 6: Developmental and Reproductive Effects [1].

Species	Exposure/Response	Reference
Rabbits, New Zealand, white, females	Exposure: Triclopyr TEA via gavage on days 6 through 18 of gestation at dose levels of 0, 10, 30, or 100 mg/kg/day. Response: Severe maternal toxicity at 100 mg/kg, including mortality, body weight loss, decreased feed consumption, increased abortions (attributed to maternal toxicity), and increased liver and kidney weights. Equivocal effects on abortions and early deliveries, associated with weight loss or anorexia in affected dams were observed at 30 mg/kg/day No developmental or teratogenic effects even at maternally toxic dose levels. Maternal NOEL = 10 mg/kg/day Developmental NOEL = 100 mg/kg/day [Abstract [1]]	Breslin and Billington 1995

[1] Summary based only on published abstract.

Appendix 7: Mutagenicity studies on triclopyr.

Organism	Exposure Level	Assay System	Effects	Reference
Salmonella typhimurium	≤5000 µg/plate	Ames assay	negative results with strains TA98 and TA100	Moriya et al. 1983
Salmonella typhimurium (TA 98, TA 100, TA 1535, and TA 1537)	triclopyr BEE (98% a.i.) 50-5000 µg/plate with or without metabolic activation	Ames assay	negative results	MRID 41732202
B. subtillis	triclopyr technical acid at 20-2000 µg/disk	recombination repair assay using rec-assay mutant (H17) and recombination repair deficient mutant (M45)	no evidence of growth inhibition for the repair competent or repair deficient bacterial strains used.	MRID 00038408
Salmonella typhimurium (TA 98 and TA 100)	triclopyr technical acid at 1-5000 µg/plate	reverse mutation assay	no increases in number of revertant colonies in the absence or presence of liver S-9 for strains tested.	MRID 00038408
Salmonella typhimurium (TA 98, TA 100, TA 1535, TA 1537 and TA 1588)	triclopyr technical (98% a.i.) at 10, 1000, 10,000 µg/plate with or without metabolic activation (rat liver S-9)	Ames assay	no significant increases in the number of revertant colonies in the absence or presence of metabolic activation	MRID 00031939
mice	triclorpyr BEE at 0, 60, 200, or 600 mg/kg	*in vivo* micronucleus assay	not clastogenic	MRID 41747101
mice, ICR, males, 10/dose group	triclopyr at single oral doses of 0, 0.7, 7.0, or 70 mg/kg followed immediately by ip injection of *Salmonella* TA-1530, *Salmonella* G-46 or *Saccharomyces* D-3	host-mediated assay	no significant increases in mutant or recombinant frequencies at any dose level, compare with controls	MRID 00057085
mice, ICR, males, 10/dose group	triclopyr at doses of 0, 0.7, 7.0, or 70 mg/kg once/day for 5 days followed by ip injection of *Salmonella* TA-1530, *Salmonella* G-46 or *Saccharomyces* D-3 on day 5	host-mediated assay	no significant increases in mutant or recombinant frequencies at any dose level, compare with controls	MRID 00057085

Appendix 7: Mutagenicity studies on triclopyr.

Organism	Exposure Level	Assay System	Effects	Reference
mice, 30 treated males per dose group each mated to 4 untreated mature virgin females	dietary levels of 0, 3, 15, or 70 mg/kg/day triclopyr (NOS) for 9 consecutive weeks	dominant lethal assay	no significant toxic effects in treated males and no significant differences in body weights; no significant effects on fertility index, average number of implantations, average resorption rate, or average litter size in any of the untreated females bred to treated males	MRID 00028996
rat	triclopyr BEE at 1.0-1000µg/mL	unscheduled DNA synthesis with rat hepatocytes	No DNA damage or inducible repair in hepatocytes	MRID 41747102
rats, Sprague-Dawley, 5/dose group	triclopyr (NOS) at single doses of 0.7, 7.0, or 70 mg/kg	*in vivo* cytogeneticis study	no cells with chromosomal aberrations observed upon sacrifice at 6, 24, or 48 hours	MRID 00057086
rats, Sprague-Dawley, 5/dose group	triclopyr at single doses of 0.7, 7.0, or 70 mg/kg for 5 days	*in vivo* cytogeneticis study	no cells with chromosomal aberrations observed upon sacrifice on day 5	MRID 00057086
rats, Sprague-Dawley males, 10/dose group, sequentially mated to 2 untreated females/week for 7 weeks	triclopyr (NOS) at 0.7, 7.0, and 70.0 mg/kg for 7 weeks 0.3 mg/kg triethylene melamine served as positive control; negative control = corn oil plus saline	dominant lethal assay	at week 1 a decrease in mating index at 7 and 70 mg/kg dose levels; trend toward increase in average number of resorptions at 7 and 70 mg/kg dose levels, with statistical significance apparent at week 4 (7 mg/kg), week 5 (70 mg/kg), and week 7 (70 mg/kg). At 70 mg/kg/, increased proportion of one or more dead implanta-tions, compared with controls	MRID 00057087

Appendix 7: Mutagenicity studies on triclopyr.

Organism	Exposure Level	Assay System	Effects	Reference
rats	$5x10^{-3}$, $1.56x10^{-3}$, $5x10^{-4}$, $1.56x10^{-4}$, $5x10^{-5}$, $1.56x10^{-5}$, $5x10^{-6}$M triclopyr for 18 hours in the presence of µCi/mL ^{3}H-thymidine	rat hepatocyte unscheduled DNA synthesis	toxicity to hepatocyte cultures, manifested as granular appearance of hepatocytes, occurred at $1.56x10^{-5}$M and increased in dose related manner until no cells remained viable at $5x10^{-3}$M. Triclopyr did not elicit significant DNA repair in primary cultures	MRID 40055702

Appendix 8: Repeated dermal dosing studies with triclopyr.

Rabbits, New Zealand white, two, weighing 2.91 and 2.95 kg	Dermal application of Garlon 4E diluted 1:1 with water at a dose equivalent to 500 mg triclopyr/kg body weight/day 5 days/week for 3 weeks (treatment amounted to 2.1 mL aqueous Garlon 4E/kg/day	Severe skin effects consisting of moderate erythema, slight edema, distinct scaliness, and slight to distinct necrosis. Autopsy indicated that treatment-related changes were in the skin only. Microscopic examination of the skin showed slight to moderate treatment-related changes. Average triclopyr recovery in the urine over days 1, 7, 14, or 21 of treatment was about 8.5%, with less recovered on day 21 than on days 1,7, and 14.	Van Beeck and Leegwater 1981a MRID 00153845
Rabbits, New Zealand white, young adult females, two/dose group, initial weights of 2.66-3.18 kg	Dermal application of Garlon 4E diluted 1:1 with water at doses equivalent to 125 or 250 mg triclopyr/kg body weight/day 5 days/week for 3 weeks (treatment amounted to 0.5 or 1.0 mL 50% aqueous Garlon 4E/kg/day)	Treatment caused moderate skin effects at low dose and moderate to severe skin effects at the high dose. Microscopic examination of the skin indicated slight to moderate treatment-related lesions. Average triclopyr recovery in the urine collected on days 1,7, 14, and 21 was about 8% at the low dose and about 9% at the high dose. There was no significant increase or decrease in triclopyr excretion during the course of the 3-week application period.	Van Beeck and Leegwater 1981b MRID 00153846

Rats, Wistar, 25 males and 25 females, 8- to 9-weeks-old, weighing 200-300 g (males) or 150-200 g (females)	Dermal application of 1 mL/kg body weight/day (i.e., daily doses of 0.05, 0.5 or 1.0 mL Garlon 4/kg body weight) 5 days/week for 3 weeks. [The Garlon 4 contained 480 mg a.i./mL. Thus, the doses corresponded to 24, 240, and 480 mg a.i./kg bw/day.] Dermal applications were made to the shaved skin area of the shoulder (surface of exposed areas was as close to 10% of the body surface as possible). Control group was treated dermally with 1 mL water/kg body weight/day	NOEL < 5% Garlon 4 (males) NOEL = 5% Garlon 4 (females) Adverse effects included: •very slight skin irritation in males at 5% dose •slight to moderate skin irritation in males and females at 50% dose •severe skin irritaiton in males and females at 100% dose •abnormal behavior in males and females at 100% dose •significant growth retardation in males at all dose levels •significantly decreased food intake in males at all dose levels •significantly decrease in food efficiency in males at all dose levels and in females at 100% dose level •histopathological changes in the skin of males and females at 50 or 100% dose levels.	Van Beeck et al. 1984 MRID 00153806
Rabbits	1000 mg/kg/day for 21 days	Increased absolute and relative liver weight in male rabbits at 1000 mg/kg/day. These effects were judged to be not of toxicological significance. NOAEL > 1000 mg/kg bw	MRID 42212701

Appendix 9. Toxicity of triclopyr to birds

Formulation	Species	Nature of Exposure	Exposure Time	Effects	Reference
Triclopyr acid, technical grade	Mallard duck (*Anas platyrhynchus*)	acute oral	NS	LD_{50} = 1698 mg/kg	MRID 40346401 Dow Chemical 1976
Triclopry (TEA) (64.7% a.i.)	Mallard duck (*Anas platyrhynchus*)	acute oral	NS	LD_{50} = 2055[a] mg/kg	MRID 40346501 Fink 1978
Triclopyr (BEE) Garlon 4	Zebra finches (*Poephila guttata*)	acute dietary	5 days treatment; 3 days observation	LD_{50} =1923 mg/kg (95% CI = 1627-2277)	Holmes et al. 1994
Garlon 4 Triclopyr (BEE) (62.9% a.i.)	Northern bobwhite quail (*Colinus virginianus*)	acute oral	single dose	LD_{50} = 849[b] mg/kg	Campbell and Lynn 1991 MRID 41902003
Triclopyr acid, (99.0% a.i.)	Mallard duck (*Anas platyrhynchus*)	subacute (5-day) dietary	NS	LC_{50} = 5620 ppm	MRID 0031249 Wildlife International 1979
Triclopyr acid, technical grade	Northern bobwhite quail (*Colinus virginianus*)	subacute (5-day) dietary	NS	LC_{50} = 2934 ppm	MRID 40346403 Dow Chemical 1976
Triclopyr acid, technical grade	Cortunix quail	subacute (5-day) dietary	NS	LC_{50} = 3272 ppm	MRID 00049638 Dow Chemical 1973
Triclopyr (TEA) (64.7% a.i.)	Mallard duck (*Anas platyrhynchus*)	subacute (5-day) dietary	NS	LC_{50} >10,000 ppm	MRID 40346502 Fink 1977
Triclopyr (TEA) (64.7% a.i.)	Northern bobwhite quail (*Colinus virginianus*)	subacute (5-day) dietary	NS	LC_{50} = 11,622 ppm	MRID 40346503 Fink 1978

Appendix 9. Toxicity of triclopyr to birds

Formulation	Species	Nature of Exposure	Exposure Time	Effects	Reference
Triclopyr (BEE) (93.% a.i.)	Mallard duck (*Anas platyrhynchus*)	subacute (5-day) dietary	NS	LC_{50} >10,000 ppm	MRID 00134179 Wildlife International 1977
Triclopyr (BEE) (96.1.% a.i.)	Mallard duck (*Anas platyrhynchus*)	subacute (5-day) dietary	NS	LC_{50} >5401 ppm	MRID 41905502 Lynn 1991
Triclopyr (BEE) (96.1% a.i.)	Northern bobwhite quail (*Colinus virginianus*)	subacute (5-day) dietary	NS	LC_{50} = 5401ppm	MRID 41905501 Lynn 1991
Triclopyr (BEE) (93% a.i.)	Northern bobwhite quail (*Colinus virginianus*)	subacute (5-day) dietary	NS	LC_{50} = 9026 ppm	MRID 00134180 Wildlife International 1978
Triclopyr (BEE) Garlon 4	Zebra finches (*Poephila guttata*)	Sublethal dietary	29 days	No significant adverse effects at 50 ppm diet, a concentration greater than the maximum expected environmental concentration resulting from forestry applications at registered rates	Holmes et al. 1994

Appendix 9. Toxicity of triclopyr to birds

Formulation	Species	Nature of Exposure	Exposure Time	Effects	Reference
DOWCO 233 (98.9% a.i.)	Mallard duck (*Anas platyrhynchus*), males and females, 6 months old	dietary concentrations of 100, 200, or 500 ppm. Food consumption was about 10% of body weight [Table 4]. Thus, doses were about 10, 20, and 50 mg/kg bw/day.	1-generation reproduction study	no symptoms of toxicity or behavioral effects at any dose level; no treatment-related mortality; slight, statistically significant ($p<0.01$) decrease in food consumption at 100 ppm dose; statistically significant decrease in body weight at 500 ppm; no statistically significant reproductive effects at any dose level, except for statistically significant ($p<0.01$) reduction in number of 14-day-old survivors as percentage of eggs set at 500 ppm dose, with reduction in number approaching statistical significance at 200 ppm	Beavers et. al. 1980 MRID 00031250
Triclopyr (Dowco 233) 99.8% pure	Mallard duck (*Anas platyrhynchus*), males and females, 6 months old, 10 males and 25 females/dose group	dietary concentrations of 100, 200, or 500 ppm Food consumption not specified.	1-generation reproduction	At 500 ppm, there were indications of toxicity and statistically significant ($p<0.01$) depression in 14-day survivors as a percentage of eggs set. NOEL = 200 ppm LOAEL = 500 ppm	Mayes 1990b MRID 92189006
Triclopyr acid (98.9% a.i.) DOWCO 233	Northern bobwhite quail (*Colinus virginianus*)	dietary concentrations of 0,100, 200, or 500 ppm. Food consumption was 7.5 to 10% of bw [Table 4].	1-generation reproduction	No statistically significant reproductive impairment at any dose level; statistically significant reduction in eggshell thickness at 200 ppm, but this effect was not observed at 100 or 500 ppm. NOEC = 100 ppm LOEC = 200 ppm	MRID 00031251 Beavers 1979

Appendix 9. Toxicity of triclopyr to birds

Formulation	Species	Nature of Exposure	Exposure Time	Effects	Reference
Triclopyr acid (99.8% a.i.) Dowco 233	Bobwhite quail (*Colinus virginianus*), 5 months old, 12 males and 24 females/dose group	dietary concentra-tions of 0,100, 200, or 500 ppm	1-generation reproduction	No treatment related reproductive or sublethal effects; statistically significant ($p<0.01$) reduction in eggshell thickness at 200 ppm, with no apparent dose-response trend.	Mayes 1990a MRID 92189005

[a] This a.i. value is from 3176 mg/kg x 64.7% formulation
[b] This a.i. value is from 1350 mg/kg x 62.9% formulation
NS = Not specified.

Appendix 10: Toxicity of triclopyr to terrestrial plants

*Greenhouse study in which the application rates of triclopyr BEE were conducted in a 2-fold geometric series of 4.4, 8.8, 17.5, 35, 70, 140, 280, 561, 1121, or 2242 g a.i./ha. The spray solutions were applied using an overhead track sprayer to evaluate the effects of triclopyr on ***percent emergence***.	Triclopyr had a significant effect on percent emergence on alfalfa, carrot, and soybean. Alfalfa was the most sensitive species to triclopyr exposure: alfalfa NOEC = 280 g a.i./ha; EC_{50} = 357 g a.i./ha carrots NOEC = 561 g a.i./ha; EC_{50} = 499 g a.i./ha corn NOEC>2242 g a.i./ha; EC_{50}>2242 g a.i./ha oats NOEC>2242 g a.i./ha; EC_{50}>2242 g a.i./ha onions NOEC>2242 g a.i./ha; EC_{50}>2242 g a.i./ha radishes NOEC>2242 g a.i./ha; EC_{50}>2242 g a.i./ha soybeans NOEC>2242 g a.i./ha; EC_{50}>2242 g a.i./ha sunflowers NOEC>2242 g a.i./ha; EC_{50}>2242 g a.i./ha tomatoes NOEC>2242 g a.i./ha; EC_{50}>2242 g a.i./ha wheat NOEC>2242 g a.i./ha; EC_{50}>2242 g a.i./ha	Schwab 1995 MRID 43650001

*Greenhouse study in which the application rates of triclopyr BEE were conducted in a 2-fold geometric series of 4.4, 8.8, 17.5, 35, 70, 140, 280, 561, 1121, or 2242 g a.i./ha. The spray solutions were applied using an overhead track sprayer to evaluate the effects of triclopyr on ***emergence shoot length***.	Alfalfa, carrot, and soybean were the most sensitive species to triclopyr for emergence shoot length. carrots NOEC = 70 g a.i./ha; EC_{50} = 341 g a.i./ha soybeans NOEC = 70 g a.i./ha; EC_{50} = 693 g a.i./ha alfalfa NOEC = 280 g a.i./ha; EC_{50} = 355 g a.i./ha onions NOEC = 280 g a.i./ha; EC_{50}>1232 g a.i./ha tomatoes NOEC = 1121 g a.i./ha; EC_{50}>2242 g a.i./ha corn NOEC>2242 g a.i./ha; EC_{50}>2242 g a.i./ha oats NOEC>2242 g a.i./ha; EC_{50}>2242 g a.i./ha radishes NOEC>2242 g a.i./ha; EC_{50}>2242 g a.i./ha sunflowers NOEC>2242 g a.i./ha; EC_{50}>2242 g a.i./ha wheat NOEC>2242 g a.i./ha; EC_{50}>2242 g a.i./ha	Schwab 1995 MRID 43650001

*Greenhouse study in which the application rates of triclopyr BEE were conducted in a 2-fold geometric series of 4.4, 8.8, 17.5, 35, 70, 140, 280, 561, 1121, or 2242 g a.i./ha. The spray solutions were applied using an overhead track sprayer to evaluate the effects of triclopyr on ***emergence shoot weight***.	Alfalfa, carrots, soybean, and onions, were the most species to triclopyr for emergence shoot weight. The emergence shoot weight data (NOEC and EC_{50} values) are summarized below: soybeans NOEC = 35 g a.i./ha; EC_{50} = 711 g a.i./ha carrots NOEC = 70 g a.i./ha; EC_{50} = 338 g a.i./ha onions NOEC = 70 g a.i./ha; EC_{50} = 471 g a.i./ha tomatoes NOEC = 140 g a.i./ha; EC_{50} = 1565 g a.i./ha alfalfa NOEC = 280 g a.i./ha; EC_{50} = 327 g a.i./ha radishes NOEC = 1121 g a.i./ha; EC_{50} = 2240 g a.i./ha corn NOEC>2242 g a.i./ha; EC_{50}>2242 g a.i./ha oats NOEC>2242 g a.i./ha; EC_{50}>2242 g a.i./ha sunflowers NOEC>2242 g a.i./ha; EC_{50}>2242 g a.i./ha wheat NOEC>2242 g a.i./ha; EC_{50}>2242 g a.i./ha	Schwab 1995 MRID 43650001

*Greenhouse study in which the application rates of triclopyr BEE were conducted in a 2-fold geometric series of 4.4, 8.8, 17.5, 35, 70, 140, 280, 561, 1121, or 2242 g a.i./ha. The spray solutions were applied using an overhead track sprayer to evaluate the effects of triclopyr on ***vegetative vigor shoot length***.	Sunflower was the most sensitive species to triclopyr for vegetative vigor shoot length data. sunflowers NOEC = 4.4 g a.i./ha; EC_{50} = 28 g a.i./ha carrots NOEC = 17.5 g a.i./ha; EC_{50} = 150 g a.i./ha tomatoes NOEC = 35 g a.i./ha; EC_{50} = 84 g a.i./ha radishes NOEC = 35 g a.i./ha; EC_{50} = 190 g a.i./ha corn soybeans NOEC = 35 g a.i./ha; EC_{50} = 197 g a.i./ha alfalfa NOEC = 70 g a.i./ha; EC_{50} = 147 g a.i./ha corn NOEC = 140 g a.i./ha; EC_{50}>2242 g a.i./ha onions NOEC = 280 g a.i./ha; EC_{50}>2242 g a.i./ha wheat NOEC = 1121 g a.i./ha; EC_{50}>2242 g a.i./ha oats NOEC>2242 g a.i./ha; EC_{50}>2242 g a.i./ha	Schwab 1995 MRID 43650001

*Greenhouse study in which the application rates of triclopyr BEE were conducted in a 2-fold geometric series of 4.4, 8.8, 17.5, 35, 70, 140, 280, 561, 1121, or 2242 g a.i./ha. The spray solutions were applied using an overhead track sprayer to evaluate the effects of triclopyr on ***vegetative vigor shoot weight***.	Sunflower was the most sensitive species to triclopyr for vegetative vigor shoot weight data. sunflowers NOEC = 4.4 g a.i./ha; EC_{50} = 29 g a.i./ha carrots NOEC = 4.4 g a.i./ha; EC_{50} = 96 g a.i./ha tomatoes NOEC = 17.5 g a.i./ha; EC_{50} = 79 g a.i./ha soybeans NOEC = 35 g a.i./ha; EC_{50} = 100 g a.i./ha radishes NOEC = 35 g a.i./ha; EC_{50} = 175 g a.i./ha corn alfalfa NOEC = 70 g a.i./ha; EC_{50} = 107 g a.i./ha corn NOEC = 140 g a.i./ha; EC_{50} = 1413 g a.i./ha onions NOEC = 280 g a.i./ha; EC_{50} = 408 g a.i./ha wheat NOEC = 561 g a.i./ha; EC_{50}>2242 g a.i./ha oats NOEC>2242 g a.i./ha; EC_{50}>2242 g a.i./ha	Schwab 1995 MRID 43650001
*Triclopyr (BEE) mixed with water and sprayed at a volume of 93.5 L/ha at herbicide rates of 0.002, 0.009, 0.03, 0.14, or 0.56 kg/ha in a laboratory sprayer. Triclopyr was applied to monocots (corn, oats, wheat, grain sorghum, and kleingrass) and to dicots (peanuts, cotton, cucumbers, and soybeans) 3 weeks after planting.	**Monocots**: at 0.56 kg/ha, phytotoxic to all monocots: causing growth inhibition and adventitious root growth in **corn**; growth inhibition, some twisting of lower stems, and adventitious root growth in **oats**; some leaf rolling and adventitious root formation in **wheat**; under-developed roots with adventitous root formation in **grain sorghum**;, and death, swollen stem bases, and adventitious and inhibited root development in **Kleingrass**. **Dicots**: at 0.56 kg/ha, extensive damage to **peanuts**, including growth inhibition, callus growth, and shortened and twisted stems; no new growth occurred in **cotton** treated with 0.14 or 0.56 kg/ha; extensive damage to **cucumbers** at ≥0.009 kg/ha; extensive damage to **soybeans** at all treatment levels.	Bovey and Meyer 1981

Triclopyr TEA, seedling germination	NOECs of 0.0002 ppm and 0.0123 ppm in sugar beat and corn, respectively, both based on radicle length	MRID 43129801 in U.S. EPA/OPP 1998a
Triclopyr TEA, seedling emergence	Corn and radish: NOEC of 0.333 lb/acre	MRID 43129801 in U.S. EPA/OPP 1998a
Triclopyr TEA, vegetative vigor	Onion: NOEC of 0.111 Sunflower: NOEC of 0.0041	MRID 43129801 in U.S. EPA/OPP 1998a

Appendix 11: Summary of field or field simulation studies on triclopyr formulations.

Application	Observations	Reference
Triclopyr (not otherwise specified) at 2.2 kg a.i./ha applied in 1983 with and without prescribed burning in 1985, 1986, and 1987. Area: Cross Timbers Experimental Range (CTER) near Stillwater, Oklahoma, 648 ha area composed of blackjack oak, post oak, red cedar, savannas, and prairies.	Increase in the population of cotton rats in all treated areas attributed to improved habitat for the cotton rat - i.e. increase in understory cover and more abundant food. This increase was more pronounced on burned areas. Decrease in numbers of rats with helminth infections in treated areas - more pronounced in areas treated with both herbicide and burning.	Boggs et al. 1991a
see description under Boggs et al. 1991a above	Prevalence of *Cuterebra* (larvae of bot flies) infestations in small mammals (white-footed deer mice, eastern woodrats, harvest mice, and cottontail rabbits) was significantly greater on unburned sites compared to burned sites. This effect could be associated with high soil temperatures during burning.	Boggs et al. 1991b
see description under Boggs et al. 1991a above	Herbaceous forage (forbs and grasses) increased after herbicide application. No effect on nutritional status of bobwhite quail.	Boren et al. 1993
*Broadcast applications of Garlon 3A (containing 3 lbs of triclopyr triethylamine salt per gallon) were made twice during the growing season to the floor of an apple orchard in Michigan. Garlon 3A was applied at an exaggerated rate of 1.65 gallons/acre (4.95 lbs a.e./acre), which is approximately 5 times the recommended rate on the label.	No triclopyr residues detected in any of the whole apple, wet pomace or dry pomace samples. No residues of triclopyr were detected in any of the juice samples. TCP was detected at a level of 0.06 ppm in one juice sample, which was attributed to the exaggerated application rates of triclopyr to the orchard floor.	Phillips and Eruick 1991 MRID 42223802

Appendix 11: Summary of field or field simulation studies on triclopyr formulations.

Application	Observations	Reference
*Single foliar application of ^{14}C-labeled triclopyr at a concentration of 2.5 ppm. Overhead spray application occurred 3 weeks prior to normal harvest and took approximately 80 minutes. The solution volume was 17.7 L and the triclopyr was applied at the equivalent of approximately 0.58 lbs a.i./acre or 647 g a.i./ha.	Single foliar application of 2.5 ppm [^{14}C]triclopyr in water equivalent to 1" of irrigation and an equivalent rate pf 0.57 lbs a.i./acre resulted in healthy leaf and fruit growth, with normal morphology. Total radioactive residues were approximately 1 ppm in treated leaves and 0.02 ppm in treated fruit. Only 1.4% of the dose was found in the leaves, and 0.06% was found in the fruit.	Yackovich and Lardie 1996a MRID 43985401
*Application of ^{14}C triclopyr (24 ppm water solution) equivalent to approximately 1 lb a.i./acre to soil in which 25-day old radish plants were growing in a greenhouse.	Residue levels in the radish roots were >5 ppm after 7 days, while soil residues were approximately 1 ppm. Residue levels in the radish leaves ere considerably lower at 0.6 ppm. The high residue levels were the result of the high application rate of the chemical equivalent to approximately 1 lb/acre. The most abundant radioactive residue in radish roots and leaves was free triclopyr. The polar conjugates were shown to hydrolyze under acid conditions liberating free triclopyr and small amounts of TCP. TMP was not detected in the radish roots or leaves.	Yackovich and Lardie 1995a MRID 43985404

Appendix 11: Summary of field or field simulation studies on triclopyr formulations.

Application	Observations	Reference
*Aerial application of triclopyr BEE at nominal rate of 8 L Garlon 4/ha (3.84 kg a.e./ha) to approximately 100 ha of forestry land in northern Ontario in August of 1987. About 3.8 km of Dora Creek containing four species of caged aquatic organism were directly oversprayed.	During application, the largest dose of triclopyr BEE was at the downstream site. The maximum concentration in the stream was 0.35 ppm, which decreased to <0.15 ppm within 15 minutes. The TWA concentration during the 12 hours of greatest exposure was 0.11 ppm. Routine water samples at the downstream site contained less than the 0.001 ppm quantitation limit for triclopyr BEE after day 1 and through the last sample taken at 1 year. TCP residues in the stream were less than the 0.05 ppm quantitation limit during the application and for the 1 year following application at all sites. No sediment samples from any site contained quantifiable residues of triclopyr BEE or TCP during the 1year the samples were taken. No statistically significant (p=0.05) differences were observed in the mortality of three species of caged aquatic organisms (yellow perch, caddisflies, and fathead minnows). At 96 hours, a statistically significant differenc ein the mortalities between control and treated sites was observed but not considered treatment related. The triclopyr degradation half-life in soil was initially about 26 days and declined over winter. Triclopyr residue half-lives in the two species of aquatic plants varied from 4 to about 10 days. Residues in terrestrial foliage declined rapidly at first and leveled off for the 30 days that samples were collected.	Fontaine 1990 MRID 41445001

Appendix 11: Summary of field or field simulation studies on triclopyr formulations.

Application	Observations	Reference
*Aerial application of Garlon 4 at the rate of 3 quarts/acre to an experimental site located near Kosciusko, Mississippi to determine the impact of a conifer release treatment on a forest environment.	No residues of triclopyr or its metabolite TCP were found in any of the water samples, sediment, or fish samples. The decline in the number of organisms per samples from the benthic invertebrates was thought to be related to natural population dynamics.	Nugent and Schotts 1990 MRID 41353201
*Garlon 4 applied using low volume ground equipment at a rate of 3.84 kg a.i./acre to a right of way near Milton, Ontario	Estimated half-lives of triclopyr over the first three half-lives is 20 days in soil and between 5 and 20 days in vegetation.	Plaumann et al. 1983 MRID 00151968
*Garlon 4 applied aerially at a rate of 3 kg a.i./ha to an 8 hectare area of forest (NOS)	Highest concentrations of triclopyr detected on day 1 after treatment in both the bare and littered covered soils. Maximum concentration in bare soil (average 3.7 ppm) decreased to 0.3 ppm after 58 days. The triclopyr concentration in the following spring was 0.4 ppm. The maximum concentration in the litter-covered soil was 3ppm, which decreased to 0.3 ppm after 58 days. The half-life of triclopyr in soil (bare and litter-covered) is between 10 and 20 days. There were no detectable levels of TCP found in the few samples that were analyzed.	Plaumann et al. 1983 MRID 00151969

Appendix 11: Summary of field or field simulation studies on triclopyr formulations.

Application	Observations	Reference
*Helicopter application of 140 L aqueous solution containing 3.57 kg (as acid equivalent a.e.) of triclopyr ester to 0.9 ha New Zealand hillside of gorse bushes and pasture grass in March 1989.	Some contamination of a stream occurred as a result of direct spray fallout into the channel. Most of the stream contamination occurred during the first major storm event when approximately 2.9 ± 1.1% of the amount of triclopyr applied (a.e.) entered the stream. Triclopyr residues in grass and underlying 10 cm of soil decayed in an approximately exponential manner yielding half-lives of 30 and 100 days, respectively. Concentrations were well below those considered to be acutely toxic to grazing animals. Soil and grass concentrations from sites at bases of the large gorse plants increased initially but decline after the first rain storm.	Wilcock et al. 1991

Appendix 11: Summary of field or field simulation studies on triclopyr formulations.

Application	Observations	Reference
*A 1:1 mixture of triclopyr TEA and picloram applied on June 21, 1992 and June 16, 1993 by helicopter at a rate of 47.5 L/ha with 1.9 L each of picloram and triclopyr in a 1:5 diesel oil emulsion to a randomized, complete block design with three treatments: controls (no treatment), treated with herbicide in 1992 only, and treated with herbicide in 1993 only. Experimental units were 13.3 ha in size. A drift retardant (38 F) and a commercial surfactant (blend of parrafin oil, polyol fatty acid esters, plyethoxylates esters, and ethoxylated alkyl aryl phosphate esters) were used. Treatments were designed to control honey mesquite (*Prosopis glandulosa*) in Texas.	No negative impact on plant and vertebrate species richness and diversity during the first 2 years after treatment when annual rainfall was 16% above average.	Nolte and Fulbright 1997
*Ground sprayer application of 2.0 kg/ha triclopyr amine or 1.6 kg/ha triclopyr ester formulations to small (4 ha) watershed in Florida.	Triclopyr residues were not detected from monitoring of streamflow and surface groundwater (<1 m deep) for 5 months after treatment.	Neary and Michael 1996

Appendix 11: Summary of field or field simulation studies on triclopyr formulations.

Application	Observations	Reference
*Aerial application of triclopyr TEA at a rate of 2.2 kg a.i./ha to cross timbers region of Central Oklahoma with and without prescribed burning . Objective of study to evaluate habitat use by male and female white-tailed deer on areas treated with herbicides, prescribed fire, and both in the cross timber.	Both sexes selected and avoided specific brush treatments throughout the year. Female deer were considerably more selective than males of human altered habitats. No clear pattern of selection or avoidance of triclopyr and tebuthiuron treatments were apparent. Variable herbicide and burn application patterns (mosaic) seems to enhance cross timbers rangeland for white-tailed deer.	Leslie et al. 1996

Appendix 11: Summary of field or field simulation studies on triclopyr formulations.

Application	Observations	Reference
*Response of competing vegetation and planted Douglas fir were studied for 10 years after six herbicide and manual release treatments in Washington and Oregon Coast Ranges. Garlon 4 helicopter application at 1.68 kg a.e./ha in diesel oil (winter: late dormant period) or Garlon 4 helicopter application at 1.40 kg a.e./ha in water (spring: early foliar period) to 2- or 3-year-old plantations of Douglas fir. Principal woody plants on the sites included shrubs (thimbleberry, salmonberry, vine maple, and trailing blackberry) and hard wood trees (red alder, big-leaf maple, and bittercherry). Principal herbaceous plants included sword-fern, pearly-everlasting, fireweed, foxglove, and bracken fern.	Treatment caused visual symptoms of injury to Douglas fir (45% of trees); 2-years after triclopyr treatment, Douglas fir growth was less than that of untreated check, suggesting the prolonged effects of herbicide injury.	Harrington et al. 1995

Appendix 11: Summary of field or field simulation studies on triclopyr formulations.

Application	Observations	Reference
*Garlon 4 thin-line individual stem (basal) application to 2 ha plot in low elevation, southern Appalachian hardwood forest in southwest VA. Treatment with herbicide intended to remove nondesirable understory woody vegetation so as to monitor the effect of treatment on relative abundance of terrestrial salamanders.	No significant difference in the relative abundance of salamanders before and after harvest on the control (p+0.788) and triclopyr (p=0.862) treatment.	Harpole and Haas 1999
*Garlon 3A (0.23 mg triclopyr TEA) in 1:1 solution with water applied at 1mL/inch via Hypo-hatchet injection or Garlon 4 (0.23 mg triclopyr BEE) in 1:13 solution with kerosene applied at 5 mL/inch via stem spray to 12-year-old upland oak stump sprout clumps in southwestern VA.	5 years after treatment, triclopyr stem spray resulted in incremental diameter growth 25% greater than unthinned controls. Triclopyr stem spray and chainsaw treatments were effective in controlling competitors and stem spray and injection required 40 and 58% less time, respectively, and cost less than chainsaw felling. The authors conclude that stem spray application of herbicides can be a cost effective alternative to chainsaw thinning for precommercial crop stem release of oak stump sprouts.	Groninger et al. 1998

Appendix 11: Summary of field or field simulation studies on triclopyr formulations.

Application	Observations	Reference
*Garlon 3A (31.8% a.e.) was applied at 2.5 mg/L to a 6-ha river plot and at 1.75 mg/L to a 4-ha cover plot using a conventional submersed application technique to control Eurasian water milfoil.	Within the river treatment plot, triclopyr concentrations (<0.01-0.41 mg/L) were less than the proposed potable water tolerance level of 0.5 mg/L 3 days after treatment and 675 m downstream of the plot, triclopyr concentrations were <0.01-0.47 mg/L within 1 day after treatment. In the cove plot, triclopyr concentrations ranged from 0.12 to 0.29 mg/L by 7 days after treatment and from <0.01 to 0.06 mg/L at 150 m downstream. Treatment reduced milfoil biomass by 99% within 4 weeks; non-target native plant biomass increased 500-1000% by 1 year post-treatment and remained significantly higher in the cover plot at 2 years post-treatment.	Getsinger et al. 1997
*Garlon 3A tank mixture [6% formulated product by volume, 0.5% LI 700 (nonionic surfactant), and 93.5% water] was applied at 5L/ha, using a hand-held backpack sprayer, to the water side of two wetland areas in late stages of purple loosestrife invasion in the State of Washington to determine the nontarget effects of treatment.	No statistically significant decreases in the survival or growth of the bioassay organisms (duckweed, *Daphnia*, or rainbow trout), and no significant decreases in the abundance of free-living aquatic invertebrates as a result of Garlon 3A application. The authors conclude from this study that Garlon 3A at the application rate used does not pose a hazard to aquatic invertebrates in wetlands in central Washington.	Gardner and Grue 1996

Appendix 11: Summary of field or field simulation studies on triclopyr formulations.

Application	Observations	Reference
Triclopyr (commercial formulation of 480 g/L, consistent with Garlon 4) applied to soil (Elkton silt loam - plowed, disced, and harrowed) at rates of 3.4, 6.7, and 10.1 kg/ha by ground sprayer. Treated in May 1988. Site in Prince George's County, MD. Different types of vegetation planted at varying periods after application.	Wheat tolerated all applications by day 8 after application (8 DAA) in terms of visual assessment of injury but yield from untreated plots was about twice that of treated plots. Kidney beans tolerated 3.4 and 6.7 kg/ha applications 82 DAA and no effect on yield was noted. Corn tolerated 3.4 6.7 and 10.1 kg/ha by 8, 47, and 82 DAA. At 3.4 kg/ha, yield was reduced to about 80% of control level. By 82 DAA, squash emerged and grew normally at 3.4 kg/ha. Earlier plantings often resulted in emergence and plant death. At 3.4 kg/ha, the yield of okra sowed 8 DAA was not effected. Potato plant fresh weights from 436 DAA of triclopyr at 3.4 kg/ha were only moderately less (6%) than untreated controls. Bananas evidence signs of injury at all application rates. After 2 years, all cites were covered by indigenous species with no apparent differences between treated and untreated cites. By this time, all crops except bananas tolerated triclopyr residues in soil.	Coffman et al. 1993
Selective foliar application of Garlon 3A at 0.6 and 6 lb a.e./acre to understory vegetation to simulate runoff form targets in an area with very porous soil, a 9% slope, clay content <0.5%.	Estimated soil half time of about 10 days with most residues in the top 10 inches of soil. Very little residue at 10-20 inches. Residues in the top 4 inches of soil at t_0 were about 0.42 ppm.	Deubert and Corte-Real 1986
Pastures treated with triclopyr at 2.2 kg/ha in June of 1983 and burned in late spring of 1985, 1986, and 1987. Stillwater, Oklahoma.	Frequency of horseweed, rosette panicgrass, and little bluestem increased with treatment due to reduction in woody overstory. Pronounced increase in the production of forbs and browse which would likely be beneficial for wildlife habitat.	Engle et al. 1991

Appendix 11: Summary of field or field simulation studies on triclopyr formulations.

Application	Observations	Reference
Triclopyr formulation not specified. Laboratory study to determine relative sorption, mobility and degradation rates of triclopyr and 2,4-D in Crowley silt loam and Perry silty clay soils in rice producing areas of AR. [See Table 1, p. 679 for soil characteristics at various depths.]	pH affected sorption and mobility, with sorption greatest and mobility lowest on subsoil with lowest pH; average half-life for triclopyr was 138 day; degradation of triclopyr more rapid at 30 C, compared with 15 C; dissipation rate of triclopyr was lowest in the soil with highest sorption rate.	Johnson et al. 1995
Triclopyr formulation not specified. Soil degradation study in crowley silt loam soil used for growing rice [see Table 1, p. 558 for soil characteristics at different depths.]	Initial soil residues of about 2.4 ppm (mg/kg soil). Soil half lives of about 10 days at 2 cm or 20 cm (silty loam soil) and about 39 days at 60 cm (silty clay loam).	Johnson and Lavy 1994
*Two foliar broadcast applications of Grandstand herbicide to rice at the maximum application rate for rice of 1pint/acre (0.375 lbs a.e./acre or 420 g a.e./ha). The study sites were located in Mississippi, California, and Texas. Applications were made in 1999.	Average dislodgeable residues ranged from 0.382 to 0.774 $\mu g/cm^2$ at day 0 sampling. After the first application, residue levels decreased to less than the limit of detection at MS and TX sites; at the CA site, average residues were 0.008 $\mu g/cm^2$ at 19 days. After the second application, residues levels decreased to below the limit of detection by day 14 at the MS and TX sites. At the CA site, residue levels were 0.007 $\mu g/cm^2$ at 14 days and between 0.001 and 0.002 (slightly greater than the limit of detection) at days 21, 28 and 35 of sampling. The calculated half-life of triclopyr dislodgeable residues ranged from 1 to 3 days.	McCormick and Robb 2000 MRID 45249901

Appendix 11: Summary of field or field simulation studies on triclopyr formulations.

Application	Observations	Reference
*Two applications of Grandstand herbicide (triethylamine salt) were made to rice during the 1994 growing season in AR and LA. The applications were made at two rice developmental stages, 3-4 leaf and ½ " intermode elongation. The application rate was 1 pint/acre (0.375 lbs a.e./acre). Water and soil samples were analyzed for residues of triclopyr, TCP, and TMP.	Water samples taken soon after application had small amounts of degradates, typically at concentrations 2- to 3-orders of magnitude less than the parent compound. Dissipation was rapid, with triclopyr first-order half-lives for the pre-flood and post-flood applications, respectively, of 7.6 and 1.8 days in AR and 2.2 and 3.4 days in LA. Corresponding values for TCP were 1.0 and .03 days in AR and 1.2 and 0.7 days in LA. Rapid water dissipation of both triclopyr and TCP is thought to have resulted primarily from photodegradation. Half-lives for TMP were 0.5 and 4.1 days in AR and 0.4 and 2.7 days in LA. Residual concentrations when the water was drained from the rice paddies were 15.1 ng/mL (triclopyr), 0.23 ng/L (TCP), and 0 ng/L (TMP) in AR. The residual concentrations in LA were 5.7 ng/L (triclopyr), 0.05 ng/L (TCP), and 0 ng/L (TMP). Triclopyr concentrations at this time represented approximately 5 and 2% of the peak concentrations observed on 0 DAT2 at the two locations, respectively. Peak concentrations of triclopyr and TCP in were soil were similar. Again, degradates were present in the soil samples soon after triclopyr application. Peak TMP concentrations were 1- to 2-orders of magnitude lower. Dissipation was slower in soil than in water, with triclopyr first-order half-lives during the two flood decline periods ranged from 2.9 to 11.7 days. During the post drainage period, triclopyr half-lives were 158 days in AR and 117 days in LA. TCP half-lives following the post-flood treatment were 36.4 days in AR and 49.5 days in LA. In the post-drainage sampling, TCP half-lives increased to 266 days in AR and 88.8 days in LA. !-year after the post-flood application, triclopyr concentrations were 0.012	Poletika and Phillips 1996 MRID 43955901

Appendix 11: Summary of field or field simulation studies on triclopyr formulations.

Application	Observations	Reference
Triclopyr BBE (probably Garlon 4 or equivalent) at rates of 0.28, 0.56, and 1.12 kg ai/ha on a pasture by backpack sprayer.	Efficacy study on the control of souther wax myrtle. The highest application rate, substantial defoliation and mortality.	Kalmbacher et al. 1993
Triclopyr (not otherwise specified) at a rate of 4.5 kg ai/ha on pine stands by backpack sprayer at monthly intervals from April to October of 1981. Location: Sierra Nevada Mountains, elev. 1300 m.	Assayed effects on various conifer species. Jeffery Pine: Severe (>60%) damage on all dates of application, and no difference in herbicide tolerance between application dates. A slight tendency for less severe effects with applications in April and may, before new leaves began the rapid phase of growth. Sugar Pine: Maximum damage after June and October applications. Minimum damage after September application. Damage highly correlated with xylem pressure potential. Red Fir: Less injury with applications in spring and most damage from applications in summer. White Fir: Most injury during summer applications with less in May and September. Douglas Fir: Most injury with applications in May and June (period of leader growth) and least injury after applications during a time of maximum water stress. High tolerance after annual growth ceased and when water stress was high.	King and Radosevich 1985

Appendix 11: Summary of field or field simulation studies on triclopyr formulations.

Application	Observations	Reference
*Triclopyr BEE (Garlon 4) in repeated exposures of 1, 3, or 6 hours at the expected environmental concentration (EEC) of 2.7 mg/L in laboratory flow-through test system (designed to mimic lotic conditions) to determine the accumulation and persistence of triclopyr BEE on natural leaf material.	Leaf material accumulated triclopyr BEE at up to a 1000 times aqueous concentrations, but residues cleared by 48-72 hours. Accumulation and persistence depended upon water depth, velocity, and exposure duration.	Kreutweiser et al. 1998
*Garlon 4 was added to semi-static microcosms designed to simulate lentic environments. In experiments to test effects of aqueous and adsorbed triclopyr BEE, detritivores were added to the microcosm 24 hours before the pesticide applications; in experiments to test effects of adsorbed triclopyr BEE alone, the invertebrates were added 24 hours after application, when the the aqueous triclopyr BEE concentrations had dissipated by >90%.	Accumulations of triclopyr BEE in leaf packs were up to 80 times aqueous concentrations and residues persisted for 4 to 5 days. In experiments to determine the effects of both aqueous and accumulated triclopyr BEE, there was no significant mortality among three species of invertebrates: stonefly (*Pteronarcys dorsata*), cranefly (*Tipula* sp.) or caddisfly (*Pycnopsyche guttifer*) in microcosms treated at or near the EEC of 2.7 mg/L In experiments to determine effects of accumulated triclopyr BEE only (insects added after aqueous concentrations declined by more than 90% and accumulated concentrations were high) there was no significant mortality of either test species: stonefly (*Pteronarcys dorsata*) or caddisfly (*Oligostomis pardalis*), even at test concentrations near 10 times the EEC.	Kreutzweiser et al. 1998.

Appendix 11: Summary of field or field simulation studies on triclopyr formulations.

Application	Observations	Reference
*Garlon 4 mixed in river water and applied (using applicators designed to deliver exponentially declining concentrations over a 6-hour period) to lower portion of outdoor stream channels. One channel was treated at an initial concentration of 2.7 mg/L (EEC) and athe other channel was treated at an initial concentration of 27 mg/L (10 times the EEC).	Accumulated triclopyr BEE in leaf material from stream channels was calculated from nominal aqueous concentrations and ranged from 55.8 to 274.4, which was clearly less than in laboratory experiments described above. The application of triclopyr BEE to the stream channels die not affect the survival of two caddisfly species, perhaps due to the rapid absorption to organic material, which mitigated the toxicity of ticlopyr BEE to the detritivores.	Kreutzweiser et al. 1998

Appendix 11: Summary of field or field simulation studies on triclopyr formulations.

Application	Observations	Reference
Field evaluation of triclopyr ester (TBEE) toxicity to trout. Lake enclosures treated by backpack application at level of 0.25 to 7.6 mg a.e./L.	Median dissipation times of 4-8 days. Cages Rainbow Trout: All rainbow trout died by day 3 at initial concentrations of 0.69-7.6 mg/L and partial mortality at 0.45 mg/L. No mortality at 0.25 mg/L. At both 0.25 and 0.45 mg/L, significant adverse effects on growth rate of surviving fish. These concentrations represent the maximum-expected concentrations in 5-- and 15 mc deep bodies of water when directly oversprayed at an application rate of 3.84 kg/ha. Native Uncaged Brook Trout: No indication of mortality or changes in population density. Some indication, however, that growth of may have been inhibited. Native Invertebrates: Only transient increase in drift. At a stream collection station 15 m downstream from the lake, the maximum measured concentration of TBEE was 0.61 mg/L, which declined to <0.05 mg/L within 40 minutes.	Kreutzweiser et al. 1995
Soil column leaching studies with loam or quartz sand with triclopyr salt (99.1%), triclopyr BEE, or Garlon 4. Water added to every column every other day to simulate 2.5 cm of precipitation.	Residues found only in top 10-cm of loam soil after 54 days. Most (85%) of triclopyr metabolized to 3,,5,6-trichloro-2-pyridinol with some (10%) formation of 2-methoxy-3,5,6-trichloropyridine. In sand, 65% of the applied triclopyr (acid) leached through column after 54 days. All TBEE leached through the sand column by day 34. Very little metabolism in sand for either compound.	Lee et al. 1986

Appendix 11: Summary of field or field simulation studies on triclopyr formulations.

Application	Observations	Reference
Leaf uptake studies of triclopyr and TBEE in wheat, barley, and chickweed.	Hydrolysis of TBEE essentially complete after 3 days and half-life TBEE was <12 hours in each species. The sensitivity of each species appeared to be best associated with the rate of metabolism of the triclopyr acid for wheat (12 hour, tolerant), barley (24 hour, moderately tolerant), and chickweed (48 hour, sensitive).	Lewer and Owen 1990
Triclopyr, ^{14}C-labeled on the pyridine ring at 2.5 mg/L in water.	Blue gill sunfish exposed for 96 hours had maximum residues in edible flesh of 0.13 mg/kg (BCF=0.0.05). The maximum whole body residue was 2.33 mg/kg (BCF $\simeq$ 1). The principal metabolites were the pyridinol and pyridine analogues.	Lickly and Murphy 1987
see description under Boggs et al. 1991a above	Oak overstory replaced by elm and eastern red cedar. Understory dominated by pioneer forbs and grasses. Effects on cotton tail rabbits examined. Compared to untreated controls, triclopyr did not influence body mass or size of rabbits and had no effect on kidney fat or relative kidney weight. A slight but statistically significant increase in relative mass of the spleen on triclopyr treated areas with or without burning compared to untreated area. This difference was not significant when compared to burned areas without herbicide treatments.	Lochmiller et al. 1995
Combination of Garlon/Tordon at 11.7 L/ha and 18.7 L/ha. Formulation of Garlon not specified. Applied along a power-line corridor in Ohio in late June of 1990.	After one year, less plant coverage relative to control. A lesser but still noticeable effect after two years. Treatment favored germination of annuals rather than perennial herbs and vines. Relatively rapid recovery of trees.	Luken et al. 1993

Appendix 11: Summary of field or field simulation studies on triclopyr formulations.

Application	Observations	Reference
see description under Boggs et al. 1991a above	Increase in population density of woodrats on triclopyr treated site compared to control site associated with an increase in forage and nest-building material. No significant differences in sex and age ratios between triclopyr and triclopyr/burn sites. No effect on reproductive activity. No effect on testes or seminal vesicle gland weights for either triclopyr and triclopyr/burn sites compared to controls. No effect of treatment on body mass or stomach content weights.	McMurry et al. 1993a
As above.	Detailed study of rat diets in treated and untreated areas. In general, forb and browse diet classes were used in accordance with availability - i.e. eastern woodrats are opportunistic feeders.	McMurry et al. 1993b
As above.	Increase in population density and reproductive activity of cotton rats. This was associated with an increase in herbaceous dicots, compared to the untreated plot. Nutritional quality of herbaceous vegetation may have been enhanced by annual burning.	McMurry et al. 1994
Hand sprayer application of triclopyr BEE at rates of 0.56, 1.12, and 2.24 kg/ha. [Whether these are acid equivalents is not specified. Assume not for risk assessment.] Area: Washington, Texas, huisache plants about 1-2 meters tall (about 800/ha) on a Bleiblerville clay.	Triclopyr BEE caused no mortality in target plant (huisache) but caused a modest reduction in canopy at the two high application rates. Grasses were favored over broadleafs at the middle dose but no effect on either was seen at the low dose.	Meyer and Bovey 1990

Appendix 11: Summary of field or field simulation studies on triclopyr formulations.

Application	Observations	Reference
Laboratory studies on washoff using Garlon 4.	When applied in a manner simulating 2.24 kg ai/ha in 28 liters of water and allowed to dry for one hour, 62% of applied triclopyr could be washed off. When allowed to dry for 2 days, only 11-17% could be washed off after simulated rains of 0.75-3.5 mm.	Michael et al. 1992
Laboratory study with soil from a mixed wood clear cut. Triclopyr, as Garlon 4) added at levels of 10, 50, 100, 500, 1000, and 5000 ppm (dry weight). Emergence of seedlings naturally occurring in the soil was monitored.	Substantial inhibition of *Rubus* spp, other dicots and monocots at all concentrations of 50 ppm and above. No substantial inhibition at 10 ppm. At levels of 500 ppm and above, no germination. The concentration of 10 ppm is essentially a NOEL and 50 ppm a FEL.	Morash and Freedman 1989
Stem injection of oak trees. Application rate as wgt/area not specified.	Soil levels of triclopyr peaked after 2 months at 2.59 mg/kg (dry weight) associated with defoliation. A lesser peak at about 1 mg/kg after 5 months, could have come from bark or small branch litterfall.	Neary et al. 1988
Laboratory efficacy study to control eurasian watermilfoil, an aquatic macrophyte. Levels of 0.25-2.5 mg a.e./L (as Garlon 3A) over time periods of 2-48 hrs.	Very little effect at any concentration for exposure periods less than 6 hours. At 0.25 mg/L, effective control was associated with exposure periods of 24 (partially effective) to 72 (very effective) hours.	Netherland and Getsinger 1992

Appendix 11: Summary of field or field simulation studies on triclopyr formulations.

Application	Observations	Reference
Subsurface application (via spray boom operated from an airboat) of triclopyr TEA (formulated as Garlon 3A) to plot A (Phelps Bay) and surface application (via boomless low-volume device) of triclopyr TEA (formulated as Garlon 3A) to plot B (Carsons Bay) of Lake Minnetonka MI in summer of 1994 to control watermilfoil. Garlon 3A was applied at a rate of 2.5 mg/L, the maximum rate indicated on the label.	Triclopyr rapidly degraded to its metabolites TCP and TMP. All three compounds dissipated rapidly from sample matrices: water, sediment, fin-fish, invertebrates, and non-target plants. There were no deaths observed in any of the seven species of fish and invertebrates that were tested. Treatment with Garlon 3A at the maximum label rate resulted in complete control of Eurasian watermilfoil at both treated sites. Native plant biomass, cover, and diversity remained higher after triclopyr treatment, compared with the untreated reference plot. Triclopyr treatments did not adversely affect water quality. After the eradication of the target species (Eurasian watermilfoil), water quality conditions generally improved, especially with respect to pH and dissolved oxygen levels.	Houtman et al. 1997a MRID 44456101
Triclopyr TEA, formulated as Garlon 3A, applied to a whole pond system to measure aquatic dissipation. Garlon 3A was applied to each of three man-made outdoor ponds (California, Texas, and Missouri) using a handgun type sprayer. Applications were made either from the pond's edge or from a boat within the pond. Garlon 3A was applied to achieve a targeted in-water concentration of 2.5 mg/L, the highest rate indicated on the particular label.	Triclopyr TEA rapidly degraded to its primary metabolites, TCP and TMP. All three compounds dissipated rapidly from sample matrices: water, sediment, and fish. Treatment did not adversely affect the water quality or the biotic community in any of the ponds. The investigators report that the results of this study are similar to the results of aquatic dissipation studies conducted in Minnesota, Washington, and Georgia and conclude that the dissipation rates for triclopyr and its metabolites will be similar throughout the continental United States.	Foster et al. 1997 MRID 44456103

Appendix 11: Summary of field or field simulation studies on triclopyr formulations.

Application	Observations	Reference
Triclopyr TEA, formulated as Garlon 3A, applied via subsurface injection using a subsurface spray boom mounted in a boat to a pond in Texas to achieve a nominal application rate of 2.5 mg/L	Triclopyr TEA rapidly degraded to its primary metabolites, TCP and TMP. All three compounds dissipated rapidly from sample matrices: water, sediment, and fish. The investigators report that the results of this study are consistent with those of previous pond dissipation studies conducted in California, Missouri, and Texas and are also similar for studies conducted in reservoir, lake, and riverine systems in Minnesota, Washington, and Georgia and conclude that the dissipation rates for triclopyr and its metabolites will be similar throughout the continental United States.	Houtman et al. 1997b MRID 44456104
*Aerial application of Garlon 3A (water soluble formulation, M-3724, containing 3 lb triclopyr as the triethylamine salt/gallon) at a rate fo 3.33 gallons/acre to approximately 14.5 acres within a watershed with a slope of 8% in West VA.	Triclopyr residues in soil on the day of application varied with the amount of tree cover and averaged undetectable in the densely wooded area, 4.4 ppm in the lightly wooded area, and 18 ppm in the open areas. Residues declined to <0.1 ppm in 166 days post treatment. Triclopyr residues collected downslope from the treated area were undetectable in soil and undetectable to <0.1 ppm in water. No TCP was found in soil, except for one sample at 28 days, which contained 0.28 ppm. TCP was not detected in water. The study concludes that no significant amount of triclopyr moved from the site of application.	McKellar and Norton 1977b MRID 40346516

Appendix 11: Summary of field or field simulation studies on triclopyr formulations.

Application	Observations	Reference
Aerial application of triclopyr salt (2.2 and 4.4 kg/ha) or triclopyr BEE (1.65-3.3 kg/ha) to Oregon brushfields on clay loam soils.	Foliar $t_{½}$s ranging from about 20 to almost 300 days, depending on formulation and type of residue (crown, browse, and litter). Average initial concentrations - including both forms as well as 2,4-D and picloram) on crowns was 44 mg/kg per kg/ha applied. Residues were lower on browse (17.1) and litter (32.1). No evidence of soil leaching from an adjacent up-slope treatment area. Soil concentrations of triclopyr (both forms) ranged from about 0.3-0.7 mg/kg at 37 days to not detectable to about 0.03 mg/kg by 325 day. Somewhat higher levels of ester than amine salt (Table V, p. 581).	Newton et al. 1990
*Aerial application of Garlon 3A (water soluble formulation, M3724, containing 3 lbs triclopyr as the triethylamine salt/gallon) at a rate of 3.33 gallons/acre (10 lbs triclopyr) to the upper portion of a watershed in West VA.	No significant movement of triclopyr from the treated watershed even with the higher levels of water during intermittent rainfalls.	McKellar and Norton 1977a MRID 40346315
Garlon 3A, 2.2 and 4.4 kg/ha by aerial application.	Vegetative hardwood and shrub cover over 1.5 meters in height virtually eliminated. Differences in height and cover were apparent at 9 years after application.	Newton et al. 1992a [NJAF 9:126]
Garlon 3A, 2.2 and 4.4 kg/ha by aerial application.	Conifers dominated over hardwoods. Some injury to conifers at the higher application rate.	Newton et al. 1992b [NJAF, 9:130]

Appendix 11: Summary of field or field simulation studies on triclopyr formulations.

Application	Observations	Reference
Triclopyr (formulation not specified) at 3.4 kg a.e./ha with a polyglycol surfactant by helicopter. A hill-pastures in western Oregon with a 34% slope and silt clay loam soils. Stream adjacent to application site. No spray boundary used.	Initial soil residues of about 0.02 mg/kg which increased on day 180 to 0.93 mg/kg, presumably due to washoff. [Kinetic data at different depths given.] Soil half time of about 75 days. Most residues in the top 15 cm of soil. Only trace amounts of metabolites detected. Stream levels peaked at 95µg/L in the first 20 hours after application. In the first significant rain after application, maximum residues were 12 µg/L. The peak level over several months was 15 µg/L.	Norris et al. 1987
Triclopyr at 10.1 kg a.e./ha with a polyglycol surfactant by hand held boom sprayer. A hill-pastures in western Oregon with a 15% slope and silt clay loam soils.	Initial triclopyr grass levels of 527 mg/kg. Levels of pyridinol and pyridine metabolites were about 0.5% and 0.02%, respectively, of those of triclopyr. Half time of < 7 days [kinetic data given] in grass. By one year after treatment, grass levels of triclopyr were about 1.3 mg/kg. Initial soil residues of about 0.55 mg/kg which peaked on day 28 to 3.1 mg/kg, presumably due to washoff. Soil half time of about 81 days. Most residues in the top 15 cm of soil. [Kinetic data at different depths given.] Only trace amounts of metabolites detected.	Norris et al. 1987 This is the same paper as the previous entry but a different site.
Triclopyr at 0.4 and 0.8 kg ai/ha by backpack sprayer to 3 cultivars of rice.	Moderate injury (primarily leaf necrosis, chlorosis, and stunting) to all three cultivars at both rates of application with a dose/dependent decrease in yield.	Pantone and Baker 1992
Triclopyr (RELEASE/TBEE) at nominal rates of 0.4, 1.26, 2.12, 2.98, and 3.84 kg a.e./ha by backpack sprayers (VMD=1089 µm) in early fall to clear-cuts in New Brunswick.	Plots assayed two growing seasons after application evidenced shallow dose/response patterns in terms of decreased crown area.	Pitt et al. 1993

Appendix 11: Summary of field or field simulation studies on triclopyr formulations.

Application	Observations	Reference
Soil adsorption studies.	Adsorption decreased as organic matter deceased and pH increased.	Pusino et al. 1994
see above description for Boggs et al. 1991a	No treatment related effects on bird density. Types of birds varied from control based on habitat preference.	Schulz et al. 1992
see description under Boggs et al. 1991a above	Greater bird density and species richness in autumn and winter on herbicide plots.	Schulz and Leslie 1992
Triclopyr BEE at 1.9 kg/ha by aerial application on whole-tree clearcut in north Maine.	Stream water draining from clearcut had peak concentration of 56 µg/L, immediately after application, and 48 µg/L after an 11-mm rain which occurred 6 days after application. Below a 450 m buffer, the highest stream concentration was 11 µg/L. Over a 298 day monitoring period, estimated losses to watershed were estimated at 0.02% of applied triclopyr.	Smith and McCormack 1988
Triclopyr at 1.9 kg/ha by aerial application on whole-tree clearcut in north Maine.	Treatment increased the concentration of nitrate and Ca in the water of moderately well drained soils and in streams. Effect is secondary to decreased vegetation.	Smith et al. 1988
Triclopyr rates of 0.03 and 0.06 kg/ha on cotton to simulate drift. Fine sandy loam soil in Mississippi.	Higher application rate decrease height of cotton when applied to pin-head square but not early-bloom. Effects not seen at lower application rate. Both application rates delayed crop maturity and lowered yield.	Snipes et al. 1991

Appendix 11: Summary of field or field simulation studies on triclopyr formulations.

Application	Observations	Reference
Garlon 4E (butoxyethanol ester, 480 g a.e./L) at nominal application rate of 3 kg/ha to sand and clay soils (slope of 7-8%) in northern Ontario.	Actual application rates estimated at 3.28 and 2.85 kg/ha on sand and clay sites, respectively. $t_{½}$ for both soil types was about 2 weeks. After four weeks, however, soil levels were less than 10% of t_0 levels and did not decline further of the 48 week observation period [about 55 µg/kg in sand and 35 µg/kg in clay]. More than 97% of the applied triclopyr remained in the top 15 cm of soil, even after heavy rains. No evidence of lateral soil transport. Very low concentrations of triclopyr in runoff water (<1µg/L) from 1-150 days after treatment.	Stephenson et al. 1990
Triclopyr at 0.3, 0.4, or 0.6 kg/ha plus X-77, a surfactant, applied to cotton.	When applied to early booting stage, there was a dose/related decrease in yields. When applied to three- to four-leaf rice, hyponasty was observed.	Street et al. 1992
see description under Boggs et al. 1991a above	Effective control of dominant overstory brush species, blackjack oak and post oak. Less effective against American elm, gum burnelia, hackberry, roughleaf dogwood, buckbrush, and eastern redcedar.	Stritzke et al. 1991
Garlon 4, aerial application at 3.67 kg a.e./ha over a forest stream.	Initial peak water levels of 0.23-0.35 mg/L as TBEE. Average concentration in stream during first 12-14 hours was 0.05-0.11 mg/L. Within 72 hours, residues were <0.001 mg/L, the limit of detection. No pyridinol residues were found at the limit of detection, 0.05 mg/L.	Thompson et al. 1991

Appendix 11: Summary of field or field simulation studies on triclopyr formulations.

Application	Observations	Reference
Triclopyr BEE (RELEASE) applied by backpack sprayer (VMD of 1089 µm and application volume of 4.32 L/min) at application rates of 0.4, 1.26, 2.12, 2.98, and 3.84 kg ai/ha to sites predominated by sugar maple with other shrub species.	Foliar half times of 1.1-1.4 days for BEE and 2.6-5.7 days for triclopyr acid.	Thompson et al. 1994
Garlon 4 directly applied to stream as a point source to yield initial concentrations in water of 0.8 and 2.7 mg/L. This was intended to mimic bodies of water 50 and 15 cm deep inadvertently sprayed with TBEE at a rate of 4 kg/ha.	Maximum concentrations of TBEE in stream water of 0.848 and 0.949 mg/L. TBEE rapidly converted to triclopyr. Periods of exposure to concentrations in excess of 0.001 mg/L were less than or equal to 120 minutes, depending on the speed of the stream flow. Invertebrate drift was increased by 3-4 fold but invertebrate abundance was not affected. Species monitored included *Plecoptera*, *Trichoptera*, *Chironemidae*, *Ceratopagonidae*, and *Tipulidae*.	Thompson et al. 1995
Triclopyr BEE at 2.3 kg a.e./ha (with surfactant and diesel oil in water) applied to sites in Idaho dominated by shinyleaf ceanothus. Silt or silty loam soil.	Initial foliar residues of 362 mg/kg with a 42% decline in one day [see Table 1, p 663 of paper for residues and kinetic data for different plant species. Also see Table 2, p. 554, for kinetic analyses].	Whistenant and McArthur 1989

Appendix 11: Summary of field or field simulation studies on triclopyr formulations.

Application	Observations	Reference
Garlon 3A applied to lake in Georgia at a rate of 2.5 mg a.e./L.	Day 0 concentration close to nominal application rate. First order $t_{½}$s of 3.3, 3.5, and 0.4 days on different plots. Variability in decay rates attributed to different hydrodynamic conditions and native vegetation. Only trace amounts of pyridinol metabolite found. Sediment residues of 0.1-0.64 mg/kg. Some bioconcentration by aquatic plants (3.3-5.7 mg/kg) with first order dissipation ($t_{½}$ of about 4 days). No detectable residues in fish (<0.1 mg/kg). The pyridinol metabolite was detected in trace quantities (<0.05 mg/kg). Minor bioconcentration in crayfish (4.87 mg/kg on day 0) with first order elimination ($t_{½}$ of about 7 days). Also some bioconcentration in clams (2.5 mg/kg on day 0) with first order elimination ($t_{½}$ of about 2 days).	Woodburn et al. 1993

Appendix 12. Toxicity of triclopyr and triclopyr formulations to fish and amphibians

Formulation	Species	Nature of Exposure	Exposure Time	Effects	Reference
FRESHWATER					
			ACID		
Triclopyr acid, technical grade (Dowco 233)	Rainbow trout (*Oncorhynchus mykiss*)	NS	acute exposure	LC_{50} = 117 ppm	MRID 00049637 Dow Chemical 1973
Triclopyr acid, technical grade (Dowco 233)	Bluegill sunfish (*Lepomis macrochirus*)	NS	acute exposure	LC_{50} = 148 ppm	MRID 00049637 Dow Chemical 1973
			TEA and TEA Formulations		
Triclopyr TEA (64.7% a.i.)	Rainbow trout (*Oncorhynchus mykiss*)	flow-through	acute exposure	LC_{50} = 613 ppm	MRID 00151956 McCarty 1978
Triclopyr TEA (47.8% a.i.) (M3724)	Rainbow trout (*Oncorhynchus mykiss*)	flow-through	acute exposure	LC_{50} = 240 ppm	MRID 00049637 Dow Chemical 1973
Triclopyr TEA (64.7% a.i.)	Bluegill sunfish (*Lepomis macrochirus*)	flow-through	acute exposure	LC_{50} = 893 ppm	MRID 00151956 McCarty 1978
Triclopyr TEA (47.8% a.i.) (M3724)	Bluegill sunfish (*Lepomis macrochirus*)	flow-through	acute exposure	LC_{50} = 471 ppm	MRID 00049637 Dow Chemical 1973
Triclopyr TEA (64.7% a.i.)	Fathead minnow (*Pimephales promelas*)	flow-through	acute exposure	LC_{50} = 947 ppm	MRID 00151956 McCarty 1978
Triclopyr TEA (44.9% a.i.)	Fathead minnow (*Pimephales promelas*)	static bioassay	acute exposure	LC_{50} = 544 ppm	MRID 00151958 Mayes 1983

Appendix 12. Toxicity of triclopyr and triclopyr formulations to fish and amphibians

Formulation	Species	Nature of Exposure	Exposure Time	Effects	Reference
Triclopyr TEA (purity NS)	Fathead minnow (*Pimephales promelas*)	static bioassay	96 hours	LC_{50} = 891 mg/L (95% CI = 787-1011)	Mayes 1990c MRID 92189012
Triclopyr TEA (44.9% a.i.)	Fathead minnow (*Pimephales promelas*)	flow-through	acute exposure	LC_{50} = 279 ppm	MRID 00151958 Mayes 1983
Triclopyr TEA Garlon 3A	Coho salmon (*Oncorhynchus kisutch*), juvenile	static	acute exposure	LC_{50} = 400 ppm	Janz et al. 1991
Garlon 3A (agri-chemical grade)	Channel catfish (*Ictalurus punctatus*)	static renewal	96 hours	LC_{50} = 344.3 ± 20.6 mg/L	Abdelghani 1995
Garlon 3A (agri-chemical grade)	Bluegill sunfish (*Lepomis macrochirus*)	static renewal	96 hours	LC_{50} = 344.3 ± 20.6 mg/L	Abdelghani 1995
Garlon 3A (31.8% **a.e**. of TEA)	Channel catfish (*I. punctatus*), juveniles, 2-3" long	static	48 hours	LC_{50} = 384.3 ± 22.0 mg Garlon 3A/L (122.6 ± 7.0 mg **a.e**./L)	Abdelghani et al. 1997
Garlon 3A (31.8% **a.e**. of TEA)	Channel catfish (*I. punctatus*), juveniles, 2-3" long	static	96 hours	LC_{50} = 344.3 ± 20.6 mg Garlon 3A/L (109.5 ± 6.6 mg **a.e**./L)	Abdelghani et al. 1997
Garlon 3A (31.4% a.e. of TEA)	Channel catfish (*Ictalurus punctatus*)	static acute toxicity	96 hours	LC_{50} = 141 mg/L triclopyr acid LC_{50} = 447 mg/L Garlon 3A	Barron and Ball 1989 MRID 41714301
Garlon 3A (31.8% **a.e**. of TEA)	Bluegill sunfish (*L. macrochirus*), juveniles, 2-3" long	static	48 hours	LC_{50} = 295.6 ± 7.6 mg Garlon 3A/L (94.0 ± 2.4 mg **a.e**./L)	Abdelghani et al. 1997
Garlon 3A (31.8% **a.e**. of TEA)	Bluegill sunfish (*L. macrochirus*), juveniles, 2-3" long	static	96 hours	LC_{50} = 286.1 ± 25.0 mg Garlon 3A/L (91.0 ± 7.9 mg **a.e**./L)	Abdelghani et al. 1997
Triclopyr (triethylamine salt)	fathead minnow	static bioassay	96 hours	LC_{50} = 245 ppm (224-269 ppm)	Mayes et al. 1984
Triclopyr (triethylamine salt)	fathead minnow	flow-through test	96 hours	LC_{50} = 120 ppm (104-140 ppm)	Mayes et al. 1984

Appendix 12. Toxicity of triclopyr and triclopyr formulations to fish and amphibians

Formulation	Species	Nature of Exposure	Exposure Time	Effects	Reference
Triclopyr (triethylamine salt)	fathead minnow	flow-through test	192 hours	LC_{50} = 101 ppm (88.5-116 ppm)	Mayes et al. 1984
Triclopyr (triethylamine salt)	fathead minnow (embryo-larval stages)	flow-through test	28 days Concentrations of 26, 43, 65, 104, 162, and 253 mg/L.	Larval survival reduced at 253 mg/L. A slight decrease in larval growth at 162 mg/L. No effects at concentrations of 104 mg/L or less.	MRID 00151958 Mayes 1983 Mayes 1990 MRID 92189012
Garlon 3A	Rainbow trout	static for lethality studies, flow through Y-maze for avoidance studies	96 hours for LC_{50}s and 0.5 hours for avoidance test	Threshold for behavioral changes = 200 ppm LC_{50} = 400 ppm Threshold for avoidance response = 800 ppm	Morgan et al. 1991
BEE and BEE Formulations					
Triclopyr BEE Garlon 4	Coho salmon (*Oncorhynchus kisutch*), juvenile	static	acute exposure	LC_{50} = 2.4 ppm	Janz et al. 1991
Garlon 4E	Rainbow trout (*Oncorhynchus mykiss*)	continuous flow	96 hours	LC_{50} = 0.8 mg/L (95% CI = 0.6-1.0)	Ross and Pell 1981 MRID 00151962
Triclopyr BEE (96.98% a.i.)	Rainbow trout (*Oncorhynchus mykiss*)	NS	acute exposure	LC_{50} = 0.65 ppm	MRID 42884501 Woodburn 1992
Triclopyr BEE (formulated.)	Rainbow trout (*Oncorhynchus mykiss*)	NS	acute exposure	LC_{50} = 1.29 ppm	MRID 00134181
Garlon 4 Triclopyr BEE (62.9% a.i.)	Rainbow trout (*Oncorhynchus mykiss*)	static	96 hours	LC_{50} = 2.6 mg/L (95% CI = 2.1-3.4)	Gorzinski et al. 1991a MRID 41971603

Appendix 12. Toxicity of triclopyr and triclopyr formulations to fish and amphibians

Formulation	Species	Nature of Exposure	Exposure Time	Effects	Reference
Garlon 4 (TSN 100516) (62.2% a.i.)	Rainbow trout (*Oncorhynchus mykiss*)	flow-through	96 hours	LC_{50} = 0.98 mg/L (95% CI = 0.824-1.18) NOEC = 0.24 mg Garlon 4/L	Weinberg et al. 1994b MRID 43442602
Triclopyr BEE (formulated.)	Bluegill sunfish (*Lepomis macrochirus*)	NS	acute exposure	LC_{50} = 1.46 ppm	MRID 00134181
Triclopyr BEE (96.98% a.i.)	Bluegill sunfish (*Lepomis macrochirus*)	NS	acute exposure	LC_{50} = 0.36 ppm	MRID 42917901 Woodburn 1993
Garlon 4 Triclopyr BEE (62.9% a.i.)	Bluegill sunfish (*Lepomis macrochirus*)	static	96 hours	LC_{50} = 1.2 mg/L (95%CI = 0.73-1.8)	Gorzinski et al. 1991b MRID 41971604
Garlon 4 (TSN 100516) (62.2% a.i.)	Bluegill sunfish (*Lepomis macrochirus*	flow-through	96 hours	LC_{50} = 0.44 mg/L (95% CI = 0.359-0.540) NOEC = 0.21 mg Garlon 4/L	Weinberg et al. 1994a MRID 43442601
Triclopyr BEE (99% a.i.)	Coho salmon (*Oncorhyncus kissutch*)	NS	acute exposure	LC_{50} = 0.45-.047 ppm (yolk-sac fry) LC_{50} = 1.4 ppm (juvenile fry)	MRID 41736304 Barron 1987
Triclopyr BEE (96.4% a.i.)	Fathead minnow (*Pimephales promelas*)	NS	24 hours	LC_{50} = 2.4 ppm	MRID 00151963 Batchelder 1980
Triclopyr BEE (96% a.i.)	Fathead minnow (*Pimephales promelas*)	NS	24 hours	LC_{50} = 2.31 ppm	MRID 00151965 Batchelder 1981
Garlon 4	Fathead minnow (*Pimephales promelas*)	static acute toxicity	96 hours	LC_{50} = 2.3 mg/L (95% CI = 2.0-2.7)	Milazzo and Batchelder 1981 MRID 00151963

Appendix 12. Toxicity of triclopyr and triclopyr formulations to fish and amphibians

Formulation	Species	Nature of Exposure	Exposure Time	Effects	Reference
Triclopyr (BEE), >99% pure	Coho salmon (*Oncorhynchus kisutch*), juveniles	static	24 hours	LC_{50} = 1.9 mg/L (95% CI = 0.78-3.6)	Barron et al. 1991
Triclopyr (BEE), >99% pure	Coho salmon (*Oncorhynchus kisutch*), juveniles	static	48 hours	LC_{50} = 1.7 mg/L (95% CI = 0.78-3.6)	Barron et al. 1991
Triclopyr (BEE), >99% pure	Coho salmon (*Oncorhynchus kisutch*), juveniles	static	96 hours	LC_{50} = 1.7 mg/L (95% CI = 0.78-3.6)	Barron et al. 1991
Triclopyr (BEE), >99% pure	Coho salmon (*Oncorhynchus kisutch*), yolk-sac fry	static	24 hours	LC_{50} = 0.72 mg/L (95% CI = 0.60-1.0)	Barron et al. 1991
Triclopyr (BEE), >99% pure	Coho salmon (*Oncorhynchus kisutch*), yolk-sac fry	static	48 hours	LC_{50} = 0.50 mg/L (95% CI = 0.36-0.60)	Barron et al. 1991
Triclopyr (BEE), >99% pure	Coho salmon (*Oncorhynchus kisutch*), yolk-sac fry	static	96 hours	LC_{50} = 0.47 mg/L (95% CI = 0.36-0.60)	Barron et al. 1991
Garlon 4	coho salmon (juvenile)	static bioassay	96 hours	LC_{50} = 2.7 ppm	Wan et al. 1991
Garlon 4	pink salmon (juvenile)	static bioassay	96 hours	LC_{50} = 1.3 ppm	Wan et al. 1991
Garlon 4	rainbow trout (juvenile)	static bioassay	96 hours	LC_{50} = 1.8 ppm	Wan et al. 1991
Garlon 4	sockeye (fingerling)	static bioassay	96 hours	LC_{50} = 1.4 ppm	Servizi et al. 1987
Garlon 4	sockeye (fry)	static bioassay	96 hours	LC_{50} = 1.2 ppm	Servizi et al. 1987
Garlon 4	rainbow trout (fry)	static bioassay	96 hours	LC_{50} = 2.2 ppm	Servizi et al. 1987
Garlon 4	coho salmon (fry)	static bioassay	96 hours	LC_{50} = 2.2 ppm	Servizi et al. 1987
Garlon 4	rainbow trout	flow-through	1 hour 6 hours 24 hours	LC_{50} = 22.5 ppm LC_{50} = 1.95 ppm LC_{50} = 0.79 ppm	Kreutzweiser et al. 1994
Garlon 4	chinook salmon	flow-through	1 hour 6 hours 24 hours	LC_{50} = 34.6 ppm LC_{50} = 4.7 ppm LC_{50} = 1.76 ppm	Kreutzweiser et al. 1994

Appendix 12. Toxicity of triclopyr and triclopyr formulations to fish and amphibians

Formulation	Species	Nature of Exposure	Exposure Time	Effects	Reference
Garlon 4	coho salmon (juvenile)	flow-through	96 hours	LC_{50} = 0.84 ppm	Johansen and Geen 1990
Garlon 4 (ethylene glycol butyl ether ester formulation)	coho salmon (juvenile)	flow-through	96 hours	lethargy occurred at concentrations >0.56 mg/L then regressed to highly distressed condition characterized by elevated oxygen uptake and death; at 0.32-0.43 mg/L fish were lethargic with reduced oxygen uptake; at concentrations ≤0.10 mg/L fish were hypersensitive to stimuli and activity levels and oxygen uptake were increased during photoperiod transitions	Johansen and Geen 1990
Garlon 4	Rainbow trout	static bioassay for LC_{50}, flow through Y-maze for avoidance studies	96 hours for LC_{50}s and 0.5 hours for avoidance test	Threshold for behavioral changes = 0.6 ppm LC_{50} = 2.4 ppm Threshold for avoidance response = 19.2 ppm	Morgan et al. 1991

See also tables in text adapted from Wan et al.1989.

Appendix 12. Toxicity of triclopyr and triclopyr formulations to fish and amphibians

Formulation	Species	Nature of Exposure	Exposure Time	Effects	Reference
MARINE					
TEA and TEA Formulations					
Triclopyr TEA (44.7% a.i.)	Tidewater silverside (*Menidia beryllina*)	acute bioassay	LC_{50} = 130 ppm	MRID 41633703 Ward 1989	
BEE and BEE Formulations					
Triclopyr BEE (96.1% a.i.)	Tidewater silverside (*Menidia beryllina*)	acute bioassay	LC_{50} = 0.45 ppm	MRID 42053901 Ward 1991	
Triclopyr BEE (62.9% a.i.) (Garlon 4)	Tidewater silverside (*Menidia beryllina*)	96-hr acute flow-through bioassay	LC_{50} = 0.77 mg/L (95% CI = 0.56-1.3)	Ward and Boeri 1991a MRID 41969901	
Triclopyr BEE (62.9% a.i.) (Garlon 4)	Tidewater silverside (*Menidia beryllina*)	acute bioassay	LC_{50} = 0.76 ppm	MRID 42053901 Ward 1991	

Appendix 13. Toxicity of triclopyr to aquatic invertebrates

Formulation, type of assay	Species	Exposure Time	Effects	Reference
Freshwater				
		ACID		
Triclopyr acid (99.5% a.i.)	Waterflea (*Daphnia magna*)	acute bioassay	LC_{50} = 132.9 ppm	MRID 40346504 McCarty 1977
Triclopyr (NOS) (>99.5%)	Waterflea (*Daphnia magna*)	static acute	LC_{50} = 132.9 mg/L	Batchelder and McCarty 1977 MRID 40346405
		TEA and TEA Formulations		
Triclopyr TEA (44.9% a.i.)	Waterflea (*Daphnia magna*)	acute bioassay	LC_{50} = 1496 ppm	MRID 00151959 Gersich 1982
Triclopyr TEA (44.9% a.i.)	Waterflea (*Daphnia magna*)	48-hr static renewal	LC_{50} = 1170 ppm (95% CI = 1030-1340)	Gersich et al. 1982 MRID 00151959
Triclopyr TEA (44.9% a.i.)	Waterflea (*Daphnia magna*)	48-hr flow-through	LC_{50} = 1110 ppm (95% CI = 980-1281)	Gersich et al. 1985 MRID 00151960
Triclopyr TEA (44.9% a.i.)	Daphnid (*Daphnia magna*)	static renewal chronic toxicity	21-day LC_{50} = 1140 mg/L (95% CI = 950-1590)	Gersich et al. 1982 MRID 00151959
Garlon 3A	Crawfish (*Procambarus spp*)	96-hr static renewal	LC_{50} = 20,117.9 ± 1,073.0 mg/L	Abdelghani 1995
Garlon 3A (31.8% **a.e**. of TEA)	Crawfish (*Procambarus spp*)	48- hr static	LC_{50} = 28,489.9 ± 1888.7 mg Garlon 3A/L (9059.8 ± 600.6 mg **a.e**./L)	Abdelghani et al. 1997
Garlon 3A (31.8% **a.e**. of TEA)	Crawfish (*Procambarus spp*)	96- hr static	LC_{50} = 20,117.9 ± 1073.0 mg Garlon 3A/L (6397.5 ± 341.2 mg **a.e**./L)	Abdelghani et al. 1997
Triclopyr (triethylamine salt), static test	cladocera (*Daphnia magna*; crustacea)	48 hours	LC_{50} = 1170 ppm (1030-1340 ppm) no animals killed at <336 ppm; all died at >2000 ppm; water pH = 7.7-8.0; temperature = 66.7-68.5°F (19.6-20.3°C)	Gersich et al. 1984
Garlon 3A (31.4% a.e. of TEA)	Red swamp crayfish (*Procambarus clarki*)	96-hr static	LC_{50}>326 mg/L	Barron et al. 1989 MRID 41736301

Appendix 13. Toxicity of triclopyr to aquatic invertebrates

Formulation, type of assay	Species	Exposure Time	Effects	Reference
Triclopyr TEA (44.9% a.i.), static renewal (3 times/week)	Daphnid (*Daphnia magna*)	life cycle test. Concentra-tions of 80.7, 149, 290, 574, and 1177 mg/L	NOEC = 80.7 ppm LOEC = 149.0 ppm affected endpoints included total young and mean brood size	MRID00151959 Gersich 1982 also in Gersich et al., 1984
BEE and BEE Formulations				
Triclopyr BEE (96.4% a.i.)	Waterflea (*Daphnia magna*)	acute bioassay	LC_{50} = 1.7 ppm (nominal conc.)	MRID00151963 Batchelder 1980
Triclopyr BEE (96.4% a.i.)	Waterflea (*Daphnia magna*)	acute bioassay	LC_{50} = 12.0 ppm	MRID00151965 Milazzo 1981
Garlon 4, static test	*Daphnia pulex*	96 hours	EC_{50} = 1.2 ppm	Servizi et al. 1987
Garlon 4	*Daphnia magna*	48-hr static	LC_{50} = 2.2 mg/L (95% CI = 1.2-3.3)	Milazzo and Batchelder 1981 MRID 00151963
Garlon 4 (TSN 100516) (62.2% a.i.)	*Daphnia magna*	48- hr flow-through	LC_{50} = 0.43 mg/L (95% CI = 0.379-0.503) EC_{50} = 0.0.35 mg/L (95% CI = 0.271-0.412) NOEC = 0.27 mg Garlon 4/L	Weinberg et al. 1994c MRID 43442603
Garlon 4	*Ameletus* sp.	96 hours	LC_{50} = 8.55 mg/L (95% CI = 3.4-13.0)	Peterson et al. 2001
Garlon 4	*Brachycentrus americanus*	96 hours	LC_{50} = 11.3 mg/L (95% CI = 9.1-13.4)	Peterson et al. 2001
Garlon 4	*Calineuria californica*	96 hours	LC_{50} = 8.1 mg/L (95% CI = 7.01-9.06)	Peterson et al. 2001
Garlon 4	*Cinygma* sp.	96 hours	LC_{50} = 20.21 mg/L (95% CI = 13.5-27.33)	Peterson et al. 2001
Garlon 4	*Psychoglypha* sp.early instar (10 mm)	96 hours	LC_{50} = 28.34 mg/L (95% CI = 24.6-31.9)	Peterson et al. 2001
Garlon 4	*Lepidostoma unicolor*	96 hours	LC_{50} = 45 mg/L (95% CI = 42.0-49.7)	Peterson et al. 2001

Appendix 13. Toxicity of triclopyr to aquatic invertebrates

Formulation, type of assay	Species	Exposure Time	Effects	Reference
Garlon 4 (units of exposure as ester), flow through system	Ephemeroptera *Heptagenia flavescens* *Isonychia sp.* *Epeorus vitrea* Plecoptera *Acroneuria abnormis* *Pteronarcys sp.* *Paragnetina sp.* *Isogenoides sp.* Trichoptera *Pycnopsyche guttifer* *Dolophilodes distinctus* (6 hr) *Hydropsyche sp.* Odonata *Ophiogomphus carolus* Diptera *Simulium sp.*	1 hour exposure, mortality assessed at 48 hours except as noted, 6 hours for *D. distinctus* because of high mortality at 24 hours.	 LC_{50} > 320 mg/L LC_{50} > 320 mg/L LC_{50} > 320 mg/L LC_{50} > 320 mg/L LC_{50} > 290 mg/L LC_{50} > 320 mg/L LC_{50} 302.9 (249-370) slope = 3.38 LC_{50} > 290 mg/L LC_{50} 61.7 (21.8-126) LC_{50} > 290 mg/Lslope = 2.22 LC_{50} > 320 mg/L LC_{50} 0.6 (0.07-1.27) slope = 1.14	Kreutzweiser et al. 1992
Garlon 4 (units of exposure as ester), artificial stream.	*Isonychia sp.*, *Epeorus vitrea*, *Hydropsyche sp.*, and *Isogenoides sp.*	1 hour exposures	320 mg/L - Significant increase in stream drift for all species except *Hydropsyche* sp. Significant increase in mortality for *Epeorus vitrea* and *Isogenoides* sp. 32 mg/L - Significant increase in stream drift only for *Isogenoides* sp. Not seen in this species at 3.2 mg/L.	Kreutzweiser et al. 1992
Garlon 4 (units of exposure as ester), artificial stream	*Dolophilodes distinctus*	1 hour exposures	Significant increase in stream drift at 3.2 mg/L but not at 0.32 mg/L.	

Salt Water or Estuarine

TEA and TEA Formulations

Appendix 13. Toxicity of triclopyr to aquatic invertebrates

Formulation, type of assay	Species	Exposure Time	Effects	Reference
Triclopyr TEA (46.09% a.i.)	Eastern Oyster (shell deposition) (*Crassostrea virginica*)	acute bioassay	LC_{50} = 130 ppm	MRID 42646101 Kowalski 1992
Triclopyr TEA (43.8% a.i.)	Eastern Oyster (embryo-larvae) (*Crassostrea virginica*)	48 hours	EC_{50} = >56 ppm 100% abnormal development at 87 ppm	MRID 42646101 Kowalski 1992
Triclopyr TEA (43.8% a.i.)	Fiddler crab (*Uca pugilator*)	acute bioassay	LC_{50}>1000 ppm	MRID 0062623 EG&G 1975
Triclopyr TEA (46.09% a.i.)	Grass shrimp (*Palaemontes pugio*)	acute bioassay	LC_{50} = 326 ppm	MRID 42646102 Kowalski 1992
Triclopyr TEA (43.8% a.i.)	Pink shrimp (*Penaeus duorarum*)	acute bioassay	LC_{50} = 895 ppm	MRID 0062623 EG&G 1975
BEE and BEE Formulations				
Ticlopyr BEE (62.9% a.i.) (Garlon 4)	Eastern Oyster (shell deposition) (*Crassostrea virginica*)	acute bioassay	LC_{50} = 0.32 ppm	MRID 41969903 Boeri 1991
Ticlopyr BEE (62.9% a.i.) (Garlon 4)	Eastern Oyster (shell deposition) (*Crassostrea virginica*)	96-hr acute flow-through bioassay	LC_{50} = 0.30 mg/L (95% CI = 0.12-0.75)	Ward and Boeri MRID 41969904
Triclopyr BEE (96.1% a.i.)	Estaurine (Grass) shrimp (*Palaemontes pugio*)	acute bioassay	LC_{50} = 2.47 ppm	MRID 41971601 Boeri 1991
Triclopyr BEE (62.4% a.i.) (Garlon 4)	Estaurine (Grass) shrimp (*Palaemontes pugio*)	96-hr acute flow-through bioassay	LC_{50} = 1.8 mg/L (95% CI = 1.6-1.9)	Ward and Boeri 1991b MRID 41969902

NS = Not specified.

Appendix 14: Toxicity of triclopyr to aquatic plants

Formulation, type of assay	Species	Exposure Time	Effects	Reference
TEA and TEA Formulations				
Triclopyr TEA (45.1% a.i.)	*Skeletonema costatum*	96 hours	EC_{50} = 6.70 ppm a.i. NOEC = 0.40 ppm a.i.	MRID 41633707 Cowgill 1987
Triclopyr TEA (45.1% a.i.)	*Lemna gibba*	14 days	EC_{50} = 8.80 ppm a.i. NOEC = 3.5 ppm a.i.	MRID 41633709 Cowgill 1987
Triclopyr TEA (45.00% a.i.)	*Lemna gibba*	14 days	EC_{50} = 11.00 ppm a.i. NOEC = 3.5 ppm a.i.	MRID 41736302 Cowgill 1988
Triclopyr TEA (45.0% a.i.)	*Anabaena flos-aquae*	96 hours	EC_{50} = 5.90 ppm a.i. NOEC = 2.0 ppm a.i.	MRID 41633706 Cowgill 1987
Triclopyr TEA (45.01% a.i.)	*Kirchneria subcapitata (Selenastrum capicornutum)*	96 hours	EC_{50} = 7.60 ppm a.i. NOEC = 11.3 ppm a.i.	MRID 41633705 Cowgill 1987
Triclopyr TEA (45.0% a.i.)	*Navicula pelliculosa*	96 hours	EC_{50} = 15.30 ppm a.i. NOEC = 8.0 ppm a.i.	MRID 41633708 Cowgill 1987
Triclopyr acid (98.8%)	*Selenastrum capricornutum*	96 hours	EC_{50} = 32.5 ppm a.i. NOEC = 7.0 ppm a.i.	MRID 41736303 Cowgill 1989
BEE and BEE Formulations				
Triclopyr BEE (61.3% a.i.)	*Kirchneria subcapitata (Selenastrum capicornutum)*	96 hours	EC_{50} = 3.40 ppm a.i. NOEC = 2.3 ppm a.i.	MRID 41633704, 42090422 Cowgill 1989
Triclopyr BEE (96.98% a.i.)	*Lemna gibba*	14 days	EC_{50} = 0.88 ppm a.i. NOEC $\leq$0.16 ppm a.i.	MRID 42719101 Milazzo 1993
Triclopyr BEE (96.98% a.i.)	*Skeletonema costatum*	96 hours	EC_{50} = 1.17 ppm a.i. NOEC = 0.209 ppm a.i.	MRID 42721103 Hughes 1993
Triclopyr BEE (96.98% a.i.)	*Anabaena flos-aquae*	96 hours	EC_{50} = 1.97 ppm a.i. NOEC =0.52 ppm a.i.	MRID 42721101 Hughes 1993
Triclopyr BEE (96.98% a.i.)	*Navicula pelliculosa*	96 hours	EC_{50} = 0.10 ppm a.i. NOEC = 0.002 ppm a.i.	MRID 42721102 Hughes 1993

Appendix 15. Toxicity of TCP to freshwater fish

Compound (Purity)	Species	Nature of Exposure	Exposure Time	Effects	Reference
TCP (99.9% a.i.)	Bluegill sunfish	acute	NS	LC_{50} = 12.5 ppm	MRID 41829003
TCP (99.9% a.i.)	Rainbow trout	acute	NS	LC_{50} = 12.6 ppm	MRID 41829004
TCP (99.7% a.i.)	Rainbow trout	acute	NS	LC_{50} = 1.5 ppm	Wan 1987[a]
TCP (99.7% a.i.)	Coho salmon	acute	NS	LC_{50} = 1.8 ppm	Wan 1987[a]
TCP (99.7% a.i.)	Chum salmon	acute	NS	LC_{50} = 1.8 ppm	Wan 1987[a]
TCP (99.7% a.i.)	Sockeye salmon	acute	NS	LC_{50} = 2.5 ppm	Wan 1987[a]
TCP (99.7% a.i.)	Chinook salmon	acute	NS	LC_{50} = 2.1 ppm	Wan 1987[a]
TCP (99.7% a.i.)	Pink salmon	acute	NS	LC_{50} = 2.7 ppm	Wan 1987[a]

Appendix 15. Toxicity of TCP to freshwater fish

Compound (Purity)	Species	Nature of Exposure	Exposure Time	Effects	Reference
TCP (99.7% a.i.)	Rainbow trout, 200 embryos, approximately 48 hours post fertilization	mean measured concentrations of 0, 0.0808, 0.134, 0.273, 0.519, 0.989, or 2.01 mg TCP/L in flow-through system with acetone as solvent. NOTE: Adverse effects were seen in the acetone control groups. Data from these groups were not used in the statistical analysis. This approach is appropriate and conservative.	early life-stages (total of 83 days)	statistically significant (α=0.05) effects like number of embryos hatched, normal larvae at hatch, time to hatch, pre-thinning survival, post-thinning survival of embryos, and overall survival were seen at ≥0.273 mg TCP/L; days to mean hatch and growth reduction (weight and length indices) were significant for larvae at 0.134 mg TCP/L. **weight**: NOEC = 0.0808 mg/L LOEC = 0.134 mg/L **length**: NOEC = 0.0808 mg/L LOEC = 0.134 mg/L **pre-thinning survival**: NOEC = 0.989 mg/L LOEC = 2.01 mg/L **number of embryos hatched**: NOEC = 0.134 mg/L LOEC = 0.273 mg/L **days to mean hatch**: NOEC = 0.0808 mg/L LOEC = 0.134 mg/L **normal larvae at hatch**: NOEC = 0.134 mg/L LOEC = 0.273 mg/L **post-thinning survival**: NOEC = 0.134 mg/L LOEC = 0.273 mg/L **overall survival**: NOEC = 0.134 mg/L LOEC = 0.273 mg/L	Marino et al. 1999 MRID 44997301

[a]Wan et al. 1987 Bull Environ. Contam. Toxicol. 39: 721-728