B9-1.0 TRICLOPYR

B9-1.1 Background Information

IUPAC: 3,5,6-trichloro-2-pyridyloxyacetic acid

CAS: [(3,5,6-trichloro-2-pyridinyl) oxy]acetic acid

CASRN: 55335-06-3

TRICLOPYR USES:

Triclopyr is a broad leaf herbicide, and has been re-registered for use on rice, pasture and rangeland, rights-of-way, forestry, and turf, including home lawns and gardens in the U.S. and Canada (U.S. EPA, 1998; PMRA 2006).

Triclopyr was the active ingredient of herbicide product Garlon 4®. It was applied at the CFB Gagetown from 1991 to 2000 (JW, 2006).

Table B9-1 Triclopyr Usage at CFB Gagetown[a]

Year	Amount of Triclopyr Applied (kg)	Total Area Treated (ha)
1991	1535.9	811.3
1992	544	248.1
1993	226.6	82.7
1995	1600.4	560.6
1996	130.2	54.0
2000	1172.88	488.7
Total	**5.2E+03**	**2.2E+03**

[a] Adapted from JW, 2006.

B9-2.0 CHEMICAL AND PHYSICAL PROPERTIES

Formula: $C_7H_4Cl_3NO_3$

Activity: Triclopyr is in the pyridine carboxylic acid class of herbicides. Triclopyr has similar activity to the auxins, which will lead to cell plasticity and nucleic acid metabolism (Antunes-Kenyon *et al.*, 2004).

Notes: Unlike similar herbicides with comparable properties, triclopyr is not known to contain dioxin impurities. Triclopyr butoxyethyl ester (BEE) is the active ingredient in the pesticide Garlon 4®. It is formulated most often in combination with kerosene and proprietary surfactants.

Structure:

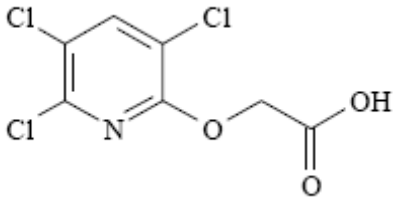

Figure B9-1 Triclopyr CASN: 5535-06-3 Structure

Table B9-2 Chemical and Physical Properties of Triclopyr

Chemical/Physical Property	Result	Reference
Colour/Form	Fluffy, colourless solid	U.S. EPA, 1998
Dissociation Constant (pKa)	3.97	JW, 2006
Henry's Law constant	$9.66x10^{-10}$ atm•m^3/mol	JW, 2006
Log K_{ow}	2.7	JW, 2006
Melting Point	148 – 150°C	U.S. EPA, 1998
Molecular Weight	256.47 g/mol	JW, 2006
Vapour Pressure	$1.26x10^{-6}$ mm Hg at 25°C	JW, 2006
Water Solubility	440 mg/L at 25°C	JW, 2006

B9-3.0 PMRA EVALUATION

The PMRA (2006) determined that triclopyr is acceptable for continued registration.

B9-4.0 TOXICOLOGICAL SUMMARY

Toxicological studies on triclopyr have been performed using the free acid, triethylamine salt (TEA) and BEE forms of triclopyr. These three forms were found to be bioequivalent for comparative tissue disposition, plasma half-life, tissue distribution, and hydrolytic cleavage under environmental and physiological conditions (U.S. EPA, 1998). With the exception of the acute toxicity studies, evaluations using all three forms of triclopyr have been included to establish values for this toxicological profile.

B9-4.1 Human Health Effects

Table B9-3 Human Health Effects Resulting from Acute Exposure of Garlon 4® Herbicide[a]

Exposure	Effects	Response
Contact	Eyes	May cause slight, temporary eye irritation. Corneal injury is unlikely
	Skin	Prolonged or repeated contact may cause skin irritation, and allergic skin reactions in some individuals. Prolonged contact may result in absorption of harmful amounts.
Ingestion	Oral	Small amounts will not likely cause any injury. Swallowing larger amounts may cause injury, induce vomiting and thus create opportunity to aspirate Garlon 4 ® into lungs during vomiting, causing lung damage or death due to chemical pneumonia.

Table B9-3 Human Health Effects Resulting from Acute Exposure of Garlon 4® Herbicide[a]

Exposure	Effects	Response
	Inhalation	May cause irritation to upper respiratory tract (nose and throat). Kerosene may cause central nervous system effects.
	Systemic	Triclopyr BEE has effects on kidneys and the liver.

[a] Dow Agrosciences, 2006.

B9-4.2 Health Effects by Route of Exposure

Health effects were reported based on data from animal studies. The majority of values displayed in this section were adapted from U.S. EPA (1998), RED document. Other values were selected from more recent studies that were not included in the RED document.

B9-4.2.1 Oral Exposure

Table B9-4 Mammalian LD_{50} Values Resulting from Oral Exposure to Triclopyr Acid

Test Type	Test Organism (Species/Sex)	LD_{50} (mg/kg)	Reference
Acute	Rat (M)	729	PMRA, 1991
	Rat (F)	630	

Table B9-5 Mammalian Systemic Effects Resulting from Oral Exposure to Triclopyr

Test Organism (Triclopyr form)	Exposure	Dose (mg/kg/day) (Duration)	Response	Reference
Sub chronic				
Fischer 344 rats (Triclopyr acid technical 98%)	Diet	0, 5, 20, 50, or 250 for 13 weeks	Degeneration of the proximal tubules of the kidneys of male and female rats was observed in increased incidences at 20 mg/kg/day. Absolute and relative kidney weight was significantly increase in male rats at the 50 mg/kg/day dose, while relative kidney weight was increased in male and female rats at 250 mg/kg/day	Landry *et al*., 1984

Table B9-5 Mammalian Systemic Effects Resulting from Oral Exposure to Triclopyr

Test Organism (Triclopyr form)	Exposure	Dose (mg/kg/day) (Duration)	Response	Reference
Chronic				
Mice (Triclopyr acid technical)	Diet	30, 60, 120, 240, 480 for 28 days	Male mice were observed with single cell necrosis of the liver, significant increases in alkaline phosphatase, AST, and ALT, and enlargement of the liver with dark color. Centrilobular swelling and degeneration of hepatocytes were observed in a dose-dependent fashion at 120 mg/kg/day and above in male mice, along with mild increases in liver enzymes at 240 mg/kg/day.	Tsuda *et al*., 1987
Beagle dogs (Triclopyr acid technical)	Diet	0, 0.1, 0.5, 2.5 for 183 days (M) 0, 0.1, 0.5, 2.5 for 184 days (F)	A decreased rate of phenolsulfonthalein (PSP) excretion was observed in dogs receiving 2.5mg/kg/day triclopyr.[a]	Quast *et al*., 1977
Beagle dogs (M/F) 14 months of age (Triclopyr acid technical)	Diet	0, 5, 10, 20 for 228 days	Decreased body weight gain in male dogs, decreased hematological parameters in male dogs, changes in clinical chemistry in male and female dogs, and liver histopathology in male and female dogs	Quast *et al*., 1976
Beagle dogs (M/F) (Triclopyr acid technical 98.9%)	Diet	0, 0.5, 2.5, 5.0 for one year	Increases in urea nitrogen and creatinine were observed at all dose levels. Changes in clinical chemistry was also observed at higher plasma concentrations.	Quast *et al*., 1976

[a] Triclopyr competes with PSP for remal excretion, hence not really a relevant effect

Table B9-6 Mammalian Reproductive and Developmental Effects Resulting from Oral Exposure to Triclopyr

Test Organism (Triclopyr form)	Exposure	Dose (mg/kg/day) (Duration)	Response	Reference
Sprague-Dawley Rats (Triclopyr acid technical 99.4%)	Diet	0, 5, 25, 250 in a multi generation study	Decreased body weight and weight gains during pre-mating for males and females. Decreased mean litter size was observed as was the mean pup weight on days 1, 4, and 21 days post - partum; An increased incidence of pup deaths was also observed at 250 mg/kg/day dose level. Increased incidence of degeneration of the proximal tubules of the kidney was observed in both sexes.	Vedula *et al*., 1995

Table B9-6 Mammalian Reproductive and Developmental Effects Resulting from Oral Exposure to Triclopyr

Test Organism (Triclopyr form)	Exposure	Dose (mg/kg/day) (Duration)	Response	Reference
Time Mated Crl:CD (SD) BR VAF/Plus Female Rats (Triclopyr TEA 46.5%)	Diet	0, 30, 100, 300 on gestation days 6 through 15 inclusive	Maternal toxicity was suggested at the 300 mg/kg dose level. Increased incidence of salivation and mortality.	Bryson *et al*., 1994
			Developmental toxicity was observed at the 300 mg/kg dose level. Decreased mean fetal body weight, increased fetal and litter incidence of skeletal anomalies include reduced ossification of one or more cranial centers and sacrocaudal vertebral arches. Increase in the number of fetuses with unossified sternebrae.	
Pregnant New Zealand White Rabbit (Triclopyr BEE)	Diet	0, 10, 30, 100 on gestation days 6 through 18 inclusive	Maternal mortality was evident at the 100 mg/kg dose level.	Bryson *et al*., 1994
			Developmental toxicity was observed at 100 mg/kg dose level. Decreased total number of live fetuses, increased total fetal deaths, increased fetal incidence of additional sternebral centers, increased incidence of reduced ossification of the digital bones, and an increase in the percentage of fetuses with 13 ribs.	
Pregnant New Zealand White Rabbit (Triclopyr TEA 46.5%)	Diet	0, 10, 30, 100 on gestation days 6 through 18 inclusive	Maternal toxicity was observed at 100 mg/kg. Increased incidence of decreased body weight gains and food efficiency, and increased liver and kidney weights were observed.	Bryson *et al*., 1994
			Developmental toxicity was observed at 100 mg/kg. Reduced number of litters, reduced number of corpora lutea, reduced number of total implants, reduced total live fetuses. Increased embryonic deaths and deaths/dam, and increased pre-implantation loss.	

B9-4.2.1.1 No Observed Adverse Effect Levels

Table B9-7 Mammalian NOAELs and LOAELs Resulting from Oral Exposure to Triclopyr

Test Organism (Triclopyr form)	Effect	Value (mg/kg/day)	Endpoint	Reference
Sub-chronic				
Fischer 344 rats (Triclopyr acid technical 98%)	NOEL	5	Histopathological changes in the kidneys of male and female rats.	Landry *et al*., 1984
	LOEL	20		

Table B9-7 Mammalian NOAELs and LOAELs Resulting from Oral Exposure to Triclopyr

Test Organism (Triclopyr form)	Effect	Value (mg/kg/day)	Endpoint	Reference
Beagle dogs (Triclopyr acid technical)	NOEL	≥ 2.5	Supplementary study, does not satisfy the guideline requirement for a subchronic toxicity study in non-rodents.	Quast *et al*., 1977
	LOEL	> 2.5		
Chronic				
Mice (Triclopyr technical 98%)	NOEL (M)[a]	28.6	Decrease in body weight gains. Increase in the incidence of thymic enlargement. Blood urea nitrogen increased in cohort with increased water consumption. Increased kidney weight and urinary protein. Increased liver weight in males.	Tsuda *et al*., 1987
	NOEL (F)[b]	26.5		
	LOEL (M)	143		
	LOEL (F)	135		
Fischer 344 rats (M/F) (Triclopyr technical 98%)	NOEL (M)	12	Marginal increases in proximal tubular degeneration at 6 months.	Eisenbrandt *et al*., 1987
	NOEL (F)	36		
	LOEL (M)	36		
	LOEL (F)	--		
Beagle dogs (M/F) 14month of age (Triclopyr acid technical)	NOEL	10	Decreased body weight gain in male dogs, decreased hematological parameters in male dogs, changes in clinical chemistry in male and female dogs, and liver histopathology in male and female dogs.	Quast *et al*., 1976
	LOEL	20		

[a] M denotes male.
[b] F denotes female.

Table B9-8 Mammalian Reproductive and Developmental NOAEL and LOAEL Values Resulting from Oral Exposure to Triclopyr

Test Organism (Species)	Effect	Value (mg/kg/day)	Endpoint	Rference
Time mated Crl:CD(SD) BR VAF/Plus female rats (Triclopyr TEA 46.5%)	NOEL (m)[a]	100	Increased signs of salivation and mortality.	Bryson *et al*., 1994b
	LOEL (m)	300		
	NOEL (d)[b]	100	Decreases in mean fetal weight, increased fetal and litter incidence of skeletal anomalies, and increased fetal incidence of unossified sternebrae.	
	LOEL (d)	300		
Male and Female Sprague-Dawley Rats, 30 males/dose; 30 females /dose, (Triclopyr acid technical 99.4%)	NOEL (m)[a]	5	Increased incidence of proximal tubular degeneration in male and female P1 and P2 rats	Vedula *et al*., 1995
	LOEL (m)	25		
	NOEL (d)[b]	25	Decreased litter size, deceased body weight and weight gain, and decreased survival in the F1 and F2 litters.	
	LOEL (d)	250		
Pregnant New Zealand White Rabbit	NOEL (m)[a]	30	Maternal toxicity, mortality	Bryson *et al*., 1994a
	LOEL (m)	100		
	NOEL (d)[b]	30	Cesarean section observed decreases	

Table B9-8 Mammalian Reproductive and Developmental NOAEL and LOAEL Values Resulting from Oral Exposure to Triclopyr

Test Organism (Species)	Effect	Value (mg/kg/day)	Endpoint	Rference
(Triclopyr BEE)	LOEL (d)	100	in total live fetuses and increases in total fetal deaths, as well as the observations of increased fetal and/or litter incidence of skeletal anomalies and variants observed at this dose.	
Pregnant New Zealand White Rabbit (Triclopyr TEA 46.5%)	NOEL (m)	30	Increased maternal effects, observed with decreased body weight gains and food efficiency. Increased liver and kidney weights were also observed.	Bryson *et al*., 1994c
	LOEL (m)	100		
	NOEL (d)	30	Reduced number of litters, reduced number of corpora lutea, reduced number of total implants, reduced total live fetuses. Increased embryonic deaths and deaths/dam, and increased pre-implantation loss.	
	LOEL (d)	100		

[a] (m) denotes maternal.
[b] (d) denotes developmental.

B9-4.2.2 Dermal Exposure

All formulations of triclopyr BEE may cause skin irritation from prolonged or repeated exposure. Garlon 4® is considered a potential skin sensitizer.

Table B9-9 Mammalian Acute LD_{50} Value Resulting from Dermal Exposure to Triclopyr BEE

Test Organism (Species/Sex)	LD_{50} (mg/kg)	Reference
Acute		
Unknown	>2,000	U.S. EPA, 1998

B9-4.2.3 Inhalation Exposure

In acute inhalation studies for triclopyr acid and triclopyr BEE LC_{50}s of >2.6 and >4.8 mg/L, respectively, were determined with a low toxicity. Therefore, significant toxicity resulting from inhalation exposure is not expected (U.S. EPA, 1998).

Table B9-10 Mammalian LC_{50} Value Resulting from Inhalation Exposure to Triclopyr

Triclopyr form	Test Organism (Species/Sex)	LC_{50} (mg/L)	Reference
Acute			
Free acid	Rat	>1.84	PMRA, 1991
BEE	Unknown	>4.8	U.S. EPA, 1998

B9-4.3 Carcinogenicity

In 1995 the U.S. EPA Carcinogenicity Peer Review Committee (CPRC) classified triclopyr as a Group D chemical (not classifiable as to human carcinogenicity) (U.S. EPA, 1998). Marginally

increased incidence of mammary tumours in female rats and mice, and adrenal pheochromocytomas in the male rat were reported by the CPRC. Overall, the animal evidence was considered marginal; therefore, triclopyr was classified as a Group D chemical (U.S. EPA, 1998).

Table B9-11 Animal Carcinogenicity Data

Test Subjects (Triclopyr form)	Exposure	Dose (mg/kg/day)	Response	Reference
Mice (Triclopyr acid technical 98%)	Diet	5.55, 28.6, 143 for 95 weeks (M) 5.09, 26.5, 135 for 95 weeks (F)	Decrease in body weight gains was observed followed by an increase in the incidence of thymic enlargement in high dose male and female mice. Blood urea nitrogen was also increased in cohort with increased water consumption. In female mice, kidney weights was increased 10 to 16% at the 135 mg/kg/day dose, while urinary protein at the 135 mg/kg/day dose was also increased at week 52. Liver weight in male mice was increased by 17% at the 143 mg/kg/day dose level at week 26 only. There were no compound-related tumors observed in male mice. Female mice had a increased trend in mammary gland adenocarcinomas at $p < 0.05$.	Tsuda *et al*., 1987
Fischer 344 Rats (M/F) (Triclopyr acid technical 98%)	Diet	50 rats received dose levels of 0, 3, 12, 36 for 2 years, 10 rats received same dose levels for 6 and 10 month	There were no significant increasing trends in tumour incidence for male rats. There weresignificant pair-wise differences *vs.* control at dose levels of 3 and 12 mg/kg triclopyr in the incidence of adrenal gland benign pheochromocytomas and benign and/or malignant pheochromocytomas combined, and in the incidence of skin fibromas at 3 and 12 mg/kg, with $p < 0.05$ for all comparisons except the incidence of pheochromocytoma (benign + combined) at 12 mg/kg, ($p < 0.01$ *vs.* control). Female rats had significant increasing trends in mammary gland adenocarcinomas at $p < 0.05$ and in adenomas and/or adenocarcinomas combined at $p < 0.01$. There was a significant difference in the pair-wise comparison of the 36 mg/kg/day dose group with the controls for mammary gland adenomas and/or adenocarcinomas combined at $p < 0.05$. There were no significant pair-wise comparisons or trends for the incidence of adrenal gland pheochromocytoma in female rats.	Eisenbrandt *et al*., 1987

B9-4.4 Populations at Special Risk

No data found

B9-4.5 Toxicokinetics

After oral absorption in humans, a dose of either 0.1 or 0.5 mg/kg body weight of technical triclopyr acid will reach peak blood concentrations within 2 to 3 hours period (PMRA, 1991). Oral absorption appeared to be dose dependent in human volunteers. The higher dose of 0.5 mg/kg body weight was absorbed at a slightly faster rate than the lower dose of 0.1 mg/kg body weight. The absorption half life for either dose however, was less than 1 hour.

It was observed that 80% of the administered technical triclopyr acid in humans was excreted in the urine unchanged over a 72 hour collection period. Most of the excretion >95% occurred during the first 24 hours after dosing, while less than 1% of the administered dose was present as the metabolite 3,5,5-trichloro-2-pyridinol. Furthermore, urinary metabolites consisted primarily of the parent acid with low levels of the pyridinol metabolite and/or conjugated derivative (PMRA, 1991).

From rat studies it was found that tissue concentrations of technical triclopyr acid were highest in the plasma, kidneys, liver, adipose tissue and the gallbladder (bile). Plasma concentration values from rats indicated that the BEE form of triclopyr will be rapidly hydrolyzed to the acid in the gut prior to, or immediately after absorption. Similar patterns of absorption, distribution, excretion and metabolism of BEE form of triclopyr was observed in rats receiving both triclopyr BEE and trclopyr acid (PMRA, 1991).

Dermal Absorption

In a rabbit dermal absorption study, 1.5% of an applied dose of triclopyr acid (2 g/kg) was reported to be absorbed through the skin (Van Beek and Leegwater, 1981a,b).

In dermal application studies, emulsifiable concentrate formulation of triclopyr containing 482 g/L triclopyr BEE was used on human volunteers. It was found that the half life for the absorption through skin was 16.8±5.2 hours with a range between 11 and 23 hours. The estimated dermal absorbed dose from urinary excretions was predicted to be 2%. However, this was an underestimate (PMRA, 1991).

In another human study, 5 volunteers received 3.7 mg/kg triclopyr BEE on the forearm for a period of 8 hours. Dermal absorption from this study was calculated to be 1.65% of the applied dose (Carmichael *et al*., 1989).

B9-4-6 Exposure Limits

Table B9-12 Existing RfD Values for Triclopyr Exposure

Reference Dose (mg/kg/day)	Route of Exposure	Reference	Endpoint	Study	Reference	NOEL (mg/kg/day)	Uncertainty Factor
Acute/Short-term (1-7 days)							
0.05[b]	Oral	U.S. EPA, 2002	Increased incidence of proximal tubular degeneration in male and female 1 generation parents	2 generation reproduction study with triclopyr acid.	--	5	100
1.0[a]	Oral	U.S. EPA, 2002	Developmental toxicity.	Developmental study with triclopyr BEE.	--	100	100
Not Required	Dermal	U.S. EPA, 1998	NOEL in a 21 day dermal toxicity study was greater than 1,000 mg/kg/day	21 day dermal toxicity study in rabbits;	Nitschke *et al*., 1989; Streeter *et al*., 1987	--	--
Not Required	Inhalation	U.S. EPA, 1998	Toxicity category IV for inhalation toxicity	Acute inhalation studies in rats	Wall *et al*., 1987	--	--
Intermediate-term (7 days- Several months)							
Not Required	Dermal	U.S. EPA, 1998	NOEL in a 21 day dermal toxicity study was greater than 1,000 mg/kg/day	21 day dermal toxicity study in rabbits;	Nitschke *et al*., 1989; Streeter *et al*., 1987	--	--
Not Required	Inhalation	U.S. EPA, 1998	Toxicity category IV for inhalation toxicity	Acute inhalation studies in rats	Wall *et al*., 1987	--	--
Long-term (6 months to lifetime)							
Not Required	Inhalation	U.S. EPA, 1998	Toxicity category IV for inhalation toxicity	Acute inhalation studies in rats	Wall *et al*., 1987	--	--
0.05[a]	Oral	U.S. EPA, 1998	Proximal tubular degeneration of the kidneys	2 generation reproduction toxicity study in rats	Vedula *et al*., 1995	5	100

[a] Value reflective of general population.
[b] Value reflective of Females between 13 to 50 years old.

Acute and chronic RfDs of 0.05 mg/kg/day (U.S. EPA, 1998) were selected for the risk assessment purposes of this report. A female specific acute RfD was selected for triclopyr as it was the most conservative value. Triclopyr was applied at Gagetown between 1991 and 2000; therefore, female military personnel may have been involved in the herbicide application.

The U.S. EPA Toxicology Endpoint Selection Committee (TESC) recommended that risk assessments for short- and intermediate term dermal exposures were not required since the NOEL in a 21 day dermal toxicity study was greater than 1,000 mg/kg/day (limit dose) in rabbits (U.S. EPA, 1998).

Similarly, the TESC concluded that a separate risk assessment for the inhalation route of exposure was not required based on the placement of triclopyr in the Toxicity Category IV for inhalation (U.S. EPA, 1998).

B9-5.0 ENVIRONMENTAL FATE AND EXPOSURE

B9-5.1 Air

Technical triclopyr acid has low vapour pressure (JW, 2006) and thus volatilization would only occur to a minor extent. Furthermore, technical triclopyr acid also has a low Henry's Law constant of $9.66x10^{-10}$ atm•m^3/mol (JW, 2006). This indicates that it have little tendency to escape from an aqueous solution. Hence, triclopyr will not be expected to be found in air with the exception of potential spray drift.

B9-5.2 Water

Technical triclopyr acid has a half life of almost nine months in water, which means that it has little tendency to hydrolyze (Linders *et al*., 1994). Photolysis appears to be the main degradation pathway for technical triclopyr acid in natural waters (Woodburn *et al*., 1993; McCall *et al*., 1986). In river water, the halflife of triclopyr was determined to be 1.3 days in artificial and natural light (Ganapathy, 1997). In natural waters, oxamic acid is the main photodegradation product with low molecular-weight organic acids as minor products (Woodburn *et al*., 1993). TBEE is not soluble in water, while technical triclopyr acid is more soluble at 440 ppm. (Royal Society of Chemistry, 1990).

Table B9-13 Half-life of Triclopyr in Water

Conditions	Half-life	Reference
Photolysis	2.8 - 83.4 hours	JW, 2006
Hydrolyzation	270 days	Linders *et al*., 1994
Photolysis in river water	1.3 days	Ganapathy, 1997

B9-5.3 Sediment and Soil

Microbial degradation is the major route of triclopyr dissipation in soil. Microbial activity and degradation will increase with temperature and moisture (Ganapathy, 1997). The aerobic degradation of technical triclopyr acid in soil will produce the metabolites 3,5,6-trichloro-2-pyridinol (TCP), 3,5,6-trichloro-2-methoxypyridine (TMP) and carbon dioxide (CO_2)(Cryer *et*

al., 1993) . The ratio of these metabolite produced from a 54 day soil column study are as follows: 4% unchanged triclopyr acid, 88% TCP, and 6% triclopyr for triclopyr acid treated soil column; 88% TCP and 7% TMP for TBEE treated soil (Lee *et al*., 1986). Triclopyr can also be degraded through soil photolysis, but this presents as a minor route of dissipation (Swann *et al.*, 1981). Triclopyr BEE has a higher tendency to adsorb to organic matter and is relatively immobile. Triclopyr acid has a similar mobility pattern as 2,4-D in soil and should be technically classified as mobile (Hamaker, 1975). However, triclopyr acid sorption to soil increases with time, thus decreases its potential for leaching (Buttler *et al*., 1993). Furthermore, the aerobic metabolites of triclopyr acid are also relatively immobile. TMP is classified as "very slightly mobile" and TCP is classified as "slightly mobile" (Linders *et al*., 1994). Most of the TCP would be expected to stay in the top one to two inches of soil (Hamaker, 1974). Triclopyr acid is degradable. However, in cold climates triclopyr acid has been reported to persist for one to two years due to a lack of microbial degradation (U.S.D.A. Forest Service, 1984). TCP is degradable but more persistent with a half-life ranging between 12 and 229 days, while TMP is the most persistent with a half life ranging from 50 to 450 days (Linders *et al*., 1984).

Table B9-14 Half-life of Triclopyr in Soil

Conditions	Half-life	Reference
--	30-90 days	JW, 2005
Field dissipation of triclopyr BEE	39 days	Ganapathy, 1997
microbial degradation	12-27 days	Linders *et al*., 1984

B9-5-4 Other Environmental Media

No data found.

B9-6.0 SUMMARY

Triclopyr is in the pyridine carboxylic acid family of herbicides. While, having similar activities as the auxins, triclopyr will alter cell plasticity and nucleic acid metabolism in plants (Antunes-Kenyon *et al*., 2004). Unlike other herbicides with comparable properties, triclopyr is not known to contain dioxin impurities. Between 1991 and 2000, approximately 5,200 kg of triclopyr was applied over an area of 2,200 ha at CFB Gagetown (JW, 2006).

Triclopyr has low acute toxicity. Adverse effects in the kidneys, and blood chemistry of rodents were observed in long-term feeding studies. Furthermore, triclopyr also caused adverse developmental/reproductive effects in rodents. The U.S. EPA (1998) has classified triclopyr as a Group D carcinogen (not classifiable as to human carcinogenicity). However, triclopyr did increase the incidence of tumours in the mammary and adrenal glands of rodents during chronic feeding studies.

B9-7.0 REFERENCES

Antunes-Kenyon, S.E., and Kennedy, G. 2004. A Review of the Toxicity and Environmental Fate of Triclopyr, Final Report: Submitted to the Massachusetts Pesticide Board Subcommittee Massachusetts Department of Agricultural Resources November 12, 2004

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. P.153. Cited In: U.S. EPA, 1998.

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. P.171. Cited In: U.S. EPA, 1998.

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. p.181. Cited In: U.S. EPA, 1998.

Buttler, I. W., Roberts, D. W., Siders, L.E. and Gardner, R.C. 1993. Non-crop right-of-way terrestrial dissipation of triclopyr in California. DowElanco. Data package Report No. ABM- 140265-E. DPR# 51566-017. Cited In: Ganapathy, 1997.

Carmichael, N.G., Nolan, R.J., Perkins, J.M., Davies, R. and Warrington, S.J. 1989. Oral and Dermal Pharmacokinetics of triclopyr in Human Volunteers. Human Toxicol. 8: 431-437. Cited In: U.S. EPA, 2002.

Cryer, S.A. *et al*. 1993. The dissipation and movement of triclopyr in a Northern U.S.A. forest ecosystem. DowElanco. Study No: PM91-2502. Data package Report No. ABM-143895-E. DPR# 51566-021. Cited In: Ganapathy, 1997.

Dow AgroSciences. Garlon 4 Herbicide. Available at: http://www.dowagro.com/PublishedLiterature/dh_0567/09002f13805673fb.pdf?filepath=ca/pdfs/noreg/010-20156.pdf&fromPage=GetDoc [Sept 26, 2006]

Eisenbrandt, D., Firchau, H., and Wolfe, E. 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. P.1094. Cited in: U.S. EPA, 1998.

Ganapathy, C. 1997. Environmental fate of triclopyr. Environmental Monitoring & Pest Management Branch, Department of Pesticide Regulation, Sacramento, CA 95814-5624. Available at: http://www.cdpr.ca.gov/docs/empm/pubs/fatememo/triclopyr.pdf [Aug 10, 2006]

Hamaker, J.W. 1975. Adsorption of triclopyr in soil. DowElanco. Data package Report No. ABM-106279-E. DPR# 51566-001. Cited In: Ganapathy, 1997.

JW. 2005. Final Report. Environmental Site Assessment of CFB Gagetown, N.B: Task 2B- Stage 1 Development of a Strategic Approach. Report to: Public Works and Government Services Canada (PWGSC) on behalf of the Department of National Defence (DND). Project No. 1002588. Jacques Whitford, December 14, 2005

JW. 2006. Final Report. Task 2A: The History and Science of Herbicide Use at CFB Gagetown From 1952 to Present. Report to: Department of National Defence. Jacques Whitford, May 19, 2006.

Landry, T., Eisenbrandt, D., and Gushow, T. 1984. Triclopyr: 13-week Dietary Toxicity in Fischer 344 Rats. Unpublished study prepared by Dow Chemical U.S.A. p.274. Cited In: U.S. EPA, 1998.

Lee, C.H., Oloffs, P.C., and Szeto, S.Y., 1986. Persistence, degradation, and movement of triclopyr and its ethylene glycol butyl ether ester in a forest soil. J. of Agriculture and Food Chem. Vol. 34. No. 6. 1075-1079. Cited In: Ganapathy, 1997.

Linders, L. B. H. J., *et al*., 1984. Pesticides:benefaction or Pandora's box? A synopsis of the environmental aspect of 243 pesticides. Research for Man and Environment. National Institute of Public Health and Environment. Bilthoven, the Netherlands. Report # 67101014. Cited In: Ganapathy, 1997.

McCall, P.J. and Gavit, P.D. 1986. Aqueous photolysis of triclopyr and its butoxyethyl ester and calculated environmental photodecomposition rates. J. of Environ. Toxicol. and Chem. Vol. 5. 879-885. Cited In: Ganapathy, 1997.

Nitschke, K., Battjes, J., Yano, B. 1989. XRM-3724. Acute Aerosol LC50 Study in Fischer 344 Rats: Lab Project No: M-003724-010. Unpublished study prepared by The Dow Chemical Co. 20 p. Cited In: U.S. EPA, 1998

PMRA. 1991. Decision Document, Triclopyr. Pesticide Management Regulatory Agency, Health Canada. Ottawa, ON.

PMRA. 2006. Re-evaluation Decision Document, Triclopyr. Pesticide Management Regulatory Agency, Health Canada. Ottawa, ON.

Quast, J.F., Humiston, C.G., LeBeau, J.E. 1976. 3, 5, 6 Trichloro-2-pyridyloxyacetic Acid (Dow co 233 Herbicide): 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-C) Cited In: U.S. EPA, 1998.

Quast, J.F., Wade, C.E., and Kalnins, R.V. 1977. 3, 5, 6-Trichloro-2-pyridinyloxyacetic Acid (Dowco 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) Cited In: U.S. EPA, 1998.

Royal Society of Chemistry. 1990. The agrochemicals handbook. Second Edition. Cambridge, England. Cited In: Ganapathy, 1997.

Streeter, C., Battjes, J., Yano, B. 1987. Triclopyr Butoxyethyl Ester, Technical: Inhalation Study in Fischer 344 Rats: Laboratory Project Study ID: K-120085-007. Unpublished study prepared by The Dow Chemical Co. 22 p. Cited In: U.S. EPA, 1998

Swann, R.L. and Unger, S.M. 1981. Photodecomposition of triclopyr butoxy ethyl ester on soil surfaces. DowElanco. Data package Report No. ABM-106279-E. DPR# 51566-001. Cited In: Ganapathy, 1997.

Tsuda, S., Ebino, K., and Ikeda, M. 1987. Triclopyr: 22-month Oral Chronic Toxicity and Oncogenicity Study in Mice. Unpublished study prepared by The Institute of Environmental Toxicology. P.1465. Cited in: U.S. EPA, 1998.

U.S. EPA. 1998. Reregistration eligibility decision for Triclopyr. U.S. Environmental Protection Agency, Prevention, Pesticides and Toxic Substances (7508C). EPA 738-R-98-011.

U.S. EPA. 2002. Triclopyr: Pesticide Tolerance. United States, Environmental Protection Agency. Fedral Register: September 18, 2002. Volume 67, Number 181.Rules and Regulations. pp.58712-58725. DOCID:fr18se02-10. Available at: http://www.epa.gov/EPA-PEST/2002/September/Day-18/p23746.htm [Oct 1, 2006]

U.S.D.A., Forest Service. 1984. Pesticide background statements. p.T1-T61. *In* Agriculture Handbook No. 633. Vol. 1. Herbicides. Part 2. Cited In: Ganapathy, 1997.

USDA/FS. 1999. Triclopyr, Herbicide Information Profile. United States Department of Agriculture, Forest Service, Pacific Northwest Region. November, 1996.

Van Beek, L. and Leegwater, D. 1981a. Study on the Percutaneous Absorption of Triclopyr by the Rabbit : Part I: Single Treatment of Abraded Skin: Report No. V81.322: Project No. B81/1735. Unpublished study prepared by Netherlands Organization for Applied Scientific Research (TNO). p. 8. Cited In: U.S. EPA, 2002.

Van Beek, L. and Leegwater, D. 1981b. Study on the Percutaneous Absorption of Triclopy by the Rabbit: Part II: Single Treatment of Intact Skin: Report No. V 81.472/212062: Project No. B 81/2062. Unpublished study prepared by Netherlands Organization for Applied Scientific Research (TNO). p. 8. Cited In: U.S. EPA, 2002.

Vedula, U., Breslin, W., and Kropscott, B. 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. P. 1065. Cited In: U.S. EPA, 1998.

Wall, J., Battjes, J., Zimmer, M. 1987. Triclopyr Butoxyethyl Ester, Technical: Acute Dermal Toxicity in New Zealand White Rabbits: Laboratory Project Study ID: K-120085-006D. Unpublished study prepared by The Dow Chemical Co. 17 p. Cited In: U.S. EPA, 1998

Woodburn, K.B., Batzer, F.R., White, F. H. and Schultz, M.R. 1993. The aqueous photolysis of triclopyr. J. of Environ. Toxicol. and Chem. Vol. 12. 43-55. Cited In: Ganapathy, 1997.