B24-1.0 2,4,5-TRICHLOROPHENOXY ACETIC ACID (2,4,5-T)

B24-1.1 Background Information

IUPAC: (2,4,5-trichlorophenoxy)acetic acid

CASRN: 93-76-5

CONTAMINANTS: 2,3,7,8 TCDD

2,4,5-T USAGE

2,4,5-T was a phenoxyacetic acid post-emergence herbicide with growth hormone-type action. 2,4,5-T was a selective herbicide widely used in crop production and in the management of forests, ranges and industrial, urban and aquatic sites. Broad-leafed plants are generally susceptible whereas most grasses, coniferous trees and certain legumes are relatively resistant. Due to significant 2,3,7,8-TCDD contamination 2,4,5-T is no longer registered for use in Canada.

2,4,5-T was used at CFB Gagetown between 1956 and 1967 (JW, 2006). 2,4,5-T was utilized for nine years to treat the range and training area (RTA) on a yearly basis; in addition, it was applied in 1966 and 1967 on designated test plots during the U.S. trials and CFS tests.

Table B24-1 2,4,5-T Usage at CFB Gagetown[a]

Year	Total 2,4,5-T Applied (kg)	Total Area Treated (ha)
1956	5,028	1,492
1957	5,290	1,570
1958	2,264	3,079
1960	8,189	3,617
1961	7,076	2,100
1963	52,023	3,706
1964	55,256	3,936
1966	418.0	51.9
1967	78.5	8.9
Total	**1.4E+05**	**2.0E+04**

[a] Adapted from Demaree and Haws, 1968; Demaree *et al*., 1966; Demaree and Creager, 1968; Boynton, 1969; and, JW, 2006.
[b] Average maximum yearly application rate (kg/ha).

B24-2.0 CHEMICAL AND PHYSICAL PROPERTIES

Formula: $C_8H_5Cl_3O_3$

Activity: herbicides (phenoxyacetic herbicides); plant growth regulators (auxins)

Notes: When this substance is used as an ester or a salt, its identity should be stated, for example 2,4,5-T-butometyl [7173-98-0], 2,4,5-T-butotyl [2545-59-7], 2,4,5-T-3-butoxypropyl [1928-48-9], 2,4,5-T-butyl [93-79-8], 2,4,5-T-2-ethylhexyl [1928-

47-8], 2,4,5-T-isobutyl [4938-72-1], 2,4,5-T-isoctyl [25168-15-4], 2,4,5-T-isopropyl [93-78-7], 2,4,5-T-methyl [1928-37-6], 2,4,5-T-pentyl [120-39-8], 2,4,5-T-sodium [13560-99-1], 2,4,5-T-triethylammonium [2008-46-0], 2,4,5-T-trolamine [3813-14-7].

Structure:

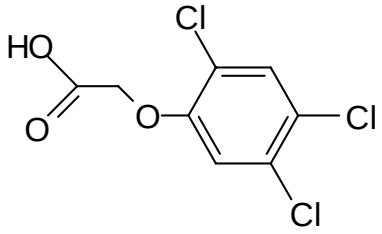

Figure B24-1 2,4,5-T CASRN 93-76-5

Table B24-2 Chemical and Physical Properties of 2,4,5-T

Chemical/Physical Property	Result	Reference
Colour/Form	Colorless to tan Crystalline solid	NIOSH, 1994
Dissociation Constant (pKa)	2.833	JW, 2006
	2.88 at 25°C	Que-Hee and Sutherland, 1981
Henry's Law constant	8.68×10^{-9} atm·m^3/mole	SRC, 1988; JW, 2006
	$<9.4 \times 10^{-17}$ atm·m^3/mol at 25°C	HSDB, 2005
Log K_{ow}	3.31	JW, 2006
	4	NIOSH, 2005
Melting Point	153°C	The Merck Index, 1983
	153-158°C	NIOSH, 2005
Molecular Weight	255.49 g/mol	The Merck Index, 1983; JW, 2006
Odour	Odourless	NIOSH, 1994
Specific Gravity	1.80 at 20°C	The Merck Index, 1983
Vapour Pressure	Negligible at 25°C	NIOSH, 2005
	700 nPa at 25°C	FAO, 2004
	$< 7.5 \times 10^{-7}$ mm Hg	Hartley and Kidd, 1983
	6.08×10^{-12} mm Hg at 25°C	JW, 2006
Water Solubility	300 mg/L	NIOSH, 2005
	268 mg/L at 25°C	Klopffer *et al.*, 1982
	278 mg/L at 25°C	Hartley and Kidd, 1983; JW, 2006
	280 mg/L at 25°C	Bailey *et al.*, 1968

B24-3.0 PMRA EVALUATION

In 1970, label revisions were required for this product and maximum TCDD residue limits were established in 1971; however, the limits were further reduced in 1981. Registration of the last remaining products was discontinued by the registrant in 1981. All basic manufacturers had already ceased production and several jurisdictions had banned use.

B24-4.0 TOXICOLOGY SUMMARY

B24-4.1 Human Health Effects

Table B24-3 Human Health Effects Resulting from Acute Exposure to Chlorophenoxy Compounds[a,b]

Exposure	Effects	Response
Acute	HEENT	Tachycardia; bradycardia; ECG abnormalities; asystole; other dysrhythmias; deaths have resulted from cardiorespiratory arrest
	Respiratory	Bradypnea, respiratory failure, hyperventilation or pulmonary edema can result from large ingestions
	Neurologic	Peripheral neuropathies Low dose: vertigo; headache; malaise; paresthesias High dose: muscle twitching; spasms
	Gastrointestinal	Nausea; vomiting; diarrhea; necrosis of the gastrointestinal mucosa
	Hepatic	Elevated LDH, AST and ALT
	Genitourinary	Albuminuria; porphyria; renal failure due to rhabdomyolysis
	Hematologic	Thrombocytopenia; leucopenia
	Dermatologic	Skin irritation
	Musculoskeletal	Muscle cramps; muscle rigidity; elevated creatine kinase

[a] Rumack and Hall, 2006.
[b] MEDITEXT®, 2006.

2,4,5-T is considered irritating to the eyes and the respiratory tract by NIOSH during short-term exposures (NIOSH , 2005). Long term or repeated exposure may cause toxicity to human reproduction or development (NIOSH, 2005).

A number of accidents have occurred during the manufacture of 2,4,5-T (FAO, 2004). Symptoms of exposed workers included shortness of breath, skin eruptions, loss of sensation in the extremities, fatigue and vertigo. Several cases in which a massive exposure to 2,4,5-T occurred produced symptoms such as mild to moderate episodes of nausea, headache, muscle cramps and fever. Stomach cramps, vomiting, diarrhea and blood in the stools may also occur. It has been generally accepted that most, if not all occupational illnesses associated with exposure to 2,4,5-T are a result of TCDD contamination (FAO, 2004).

Seven males exposed to a single oral dose of 2,4,5-T with less than 0.05 ppm TCDD showed no clinical signs of poisoning based on a neurological evaluation, blood pressure, pulse rate, pulmonary function, electrocardiogram, chest X-ray. In addition, urological, hematological and clinical chemistry parameters were examined (Gehring *et al*., 1973a). However, chlorophenoxy herbicides have produced contact dermatitis in humans (Doull *et al*., 1986) and inhalation have resulted in coughing and burning sensations in the naspharynx and chest (Morgan, 1982). Prolonged inhalation may cause dizziness (Morgan, 1982).

B24-4.2 Health Effects by Route of Exposure

B24-*4.2.1* *Oral Exposure*

B24-4.2.1.1 Death

Table B24-4 Mammalian LD_{50} Values Resulting from Oral Exposure to 2,4,5-T

Test Organism (Species/Sex)	LD_{50} (mg/kg)	Reference
Acute		
Mice	242	Senczuk and Pogorzelska, 1980
Mice	389	Hayes, 1982
Rat	300	Bailey and White, 1965
Rat	500	FAO, 2004; Hayes, 1982
Guinea pig	381	AAPCO, 1966; Hayes, 1982
Hamster	425	Grant, 1979
Dog	>100	FAO, 2004; Hayes, 1982

Table B24-5 Mortality Resulting from Oral Exposure to 2,4,5-T

Test Organism (Species)	Dose (mg/kg/day) (Duration)	TCDD contamination	Response	Reference
Acute				
Dog	20 (11-75 day)		Mortality (11-75 days after the beginning of dosing)	FAO, 2004
Mice (NMRI)	$\geq$ 80 (11 day)	0.1 ppm	Maternal toxicity	Frohberg, 1974

B-24-4.2.1.2 Systemic Effects

Table B24-6 Systemic Effects Resulting from Oral Exposure to 2,4,5-T

Test Organism (Species)	Dose (mg/kg) (Duration)	TCDD	Response	Reference
Acute				
Monkey	12 (18 day)		Loss of appetite; vomiting; decreased body weight	Dougherty *et al*., 1975
Sub-Chronic				
Rat	100 (90 day)	< 1 mg/kg	Toxic symptoms (depression in body weight, decreased food intake)	FAO, 2004
Chronic				
Rat (Sprague Dawley)	$\geq$ 10 (2 years)	< 0.04 ppb	Increase in mineralization in renal papillae or renal pelvis (F)	Kociba *et al*., 1979
Rat (Sprague Dawley)	30 (2 years)	< 0.04 ppb	Decreased body weight (F); Increased urine volume, coproporphyrin and uroporphyrin excretion (M); increased pigmentation and in proximal convoluted tubular epithelium (M); decreased spontaneous chronic nephritis; increased mineralization of pulmonary alveoli, myocardium, myocardial blood vessels, gastric mucosa and muscles; changes in liver and lungs	Kociba *et al*., 1979

(M) Effects observed in males only.
(F) Effects observed in females only.

B24-4.2.1.3 Neurological Effects

No data found.

B24-4.2.1.4 Reproductive/Developmental Effects

Table B24-7 Reproductive and Developmental Effects resulting from Oral Exposure to 2,4,5-T

Test Organism (Species)	Dose (mg/kg/day) (Duration)	TCDD	Response	Reference
Mice (NMRI)	45 (days 6 through 15 of gestation)	<0.02 ppm	Increased cleft palate induction rate	Neubert and Dillman, 1972
Mice (NMRI)	60 (days 6 through 15 of gestation)	0.055 ppm	Increased cleft palate induction rate	Neubert and Dillman, 1972
Mice (NMRI)	≥ 80[a] (days 6 through 15 of gestation)	0.1 ppm	Fetal malformations; fetal loss	Frohberg, 1974
Mice (NMRI)	120[b] (days 6 through 15 of gestation)	0.1 ppm	Fetal malformations; fetal loss	Frohberg, 1974
Rat (Sprague Dawley)	10 (multi-generation)	<0.03 ppb	Reduced fertility	Smith *et al*., 1981
Rat (Sprague Dawley)	≥10 (multi-generation)	<0.03 ppb	Decreased post-natal survival	Smith *et al*., 1981
Rat (Sprague Dawley)	30 (multi-generation)	<0.03 ppb	Increased relative liver weights; decreased thymus weight (M)	Smith *et al*., 1981

[a] 2,4,5-T free acid.
[b] 2,4,5-T ester.

B24-4.2.1.5 No Observed Adverse Effect Levels

Table B24-8 NOAELs and LOAELs for Oral Exposure to 2,4,5-T[a]

Test Organism (Species)	Effect	Value	TCDD	Endpoint	Reference
Sub-chronic					
Dog	NOAEL	10 mg/kg/day		Mortality	Drill and Hiratzka, 1953
Chronic					
Rat (Sprague Dawley)	NOAEL	30 mg/kg/day	<0.04 ppb	Food consumption; mortality; hematology; clinical chemistry	Kociba *et al*., 1979
Rat (Sprague Dawley)	NOAEL	30 mg/kg/day	0.05 ppm	Systemic toxicity	Leuschner *et al*., 1979

[a] Obtained from U.S. EPA, 1989; NIOSH, 1996; HSDB, 2005; WHO, 1979.

Table B24-9 2,4,5-T Reproductive and Developmental NOAEL and LOAEL Values[a]

Test Organism (Species)	Effect	Value (mg/kg/day)	TCDD contamination	Endpoint	eference
Mice (NMRI)	NOAEL	30	<0.02 ppm	Increased cleft palate induction rate	Neubert and Dillman, 1972
Mice (NMRI)	NOAEL	30	0.055 ppm	Increased cleft palate induction rate	Neubert and Dillman, 1972
Rat (Sprague Dawley)	NOAEL	30	<0.03 ppb	Pre-mating parental body weight, food intake or behaviour; fertility; liter size	Smith *et al*., 1981
Rat (Sprague Dawley)	NOAEL	30	<0.05 ppm	Maternal toxicity; Developmental or Reproductive toxicity	Leuschner *et al*., 1978
Rabbit (New Zealand white)	NOAEL	40	0.5 ppm	Reproductive toxicity; Fetal malformations	Emerson *et al*., 1971
Sheep	NOAEL	113	1 ppm	Fetal toxicity or malformations	Benns and Balls, 1971
Monkey (Rhesus)	NOAEL	10	0.05 ppm	Maternal and reproductive toxicity; Fetal toxicity and malformations	Dougherty *et al*., 1975

[a] Obtained from WHO, 1979; WHO, 1981.

B24-4.2.2 Dermal Exposure

Based on LD_{50} values greater than 5,000 mg/kg bw 2,4,5-T, the dimethylamine salt and the amyl ester, mixed butyl ester and 2-ethylhexyl ester were found to be of low toxicity after dermal application (DFG, 1982).

B24-4.2.2.1 Death

Table B24-10 Mammalian acute LD_{50} Value Resulting from Dermal Exposure to 2,4,,5-T

Test Type	Test Organism (Species/Sex)	LD_{50} (mg/kg)	Reference
Acute	Rat	>5,000	FAO, 2004

B24-4.2.2.2 Systemic Effects

No data found.

B24-4.2.2.3 Neurological Effects

No data found.

B24-4.2.2.4 Reproductive/Developmental Effects

No data found.

B24-4.2.2.5 No Observed Adverse Effect Level

No data found.

B24-4.2.3 *Inhalation Exposure*

Male and female rats were exposure to aerosols of 2,4,5-T and two 2,4,5-T esters at the highest possible concentrations (830, 1,200 and 1,100 mg/m^3, respectively). No deaths occurred, and only a transient decline in well-being was observed (DFG, 1982).

B24-4.2.3.1 Death

No data found.

B24-4.2.3.2 Systemic Effects

No data found.

B24-4.2.3.3 Neurological Effects

No data found.

B24-4.2.3.4 Reproductive/Developmental Effects

When mice were exposed to 2,4,5-T diethylamine salt during days 6 to 15 of gestation, high maternal toxicity, embryotoxicity and foetotoxicity was observed at 374 mg/m^3. At lower exposure concentrations (87 and 219 mg/m^3) foetal weights decreased and an increase in resorptions and cleft palate was observed (Celamerck, 1975). Exposure to 2,4,5-T butoxy ethyl ester during the same time period resulted in maternal mortality and an increase in abortions and resorptions at the highest exposure concentration (374 mg/m^3). Fetal effects were observed at lower exposure concentrations (85 and 216 mg/m^3) (Celamerck, 1974).

B24-4.2.3.5 No Observed Adverse Effect Level

No data found.

B24-4.3 Carcinogenicity

Chlorophenoxy herbicides have been classified as Group 2B: possibly carcinogenic to humans by IARC (1987). This is based on limited evidence for carcinogenicity of chlorophenoxy herbicides to humans and inadequate evidence for 2,4,5-T carcinogenicity to animals. All of the animal studies were inadequate due to the small numbers of animals used (Table B24-11) (IARC, 1987).

2,4,5-T without TCDD contamination (LOD: 0.12 to 0.033 ppb) bas been found not to be carcinogenic (FAO, 2004). Furthermore, the American Conference of Governmental Industrial Hygienists TLVs and BEIs (2005) determined that 2,4,5-T is not classifiable as a human carcinogen.

Table B24-11 Animal Carcinogenicity Data[a]

Test Subjects	Exposure	Dose	TCDD contamination	Response	Reference
Mice	Oral	--	<0.05 mg/kg	Increased incidence of tumours at various sites	IARC, 1977
Mice (XVII/G)	Oral	80 ppm	<0.05 ppm	No tumourigenic effects	Muranyi-Kovacs *et al*., 1976
Mice (C3Hf)	Oral	80 ppm	<0.05 ppm	Increased total yield of tumours	Muranyi-Kovacs *et al*., 1976
Rat	Oral	--		Increased incidence of interfollicular C-cell adenomas of the thyroid was not dose-related; no carcinogenic effects	Kociba *et al*., 1979

[a] Obtained from IARC, 1977; U.S. EPA, 1989; WHO, 1979.

B24-4.4 Populations at Special Risk

No data found.

B24-4.5 Toxicokinetics

B24-4.5.1 Absorption

Residues of 2,4,5-T in treated food or feed crops were readily absorbed in the gut of animals (Leng, 1977). Oral doses of 2,4,5-T were readily absorbed in humans (IARC, 1977).

Dermal Absorption

Exposure of humans to phenoxy acids through spraying indicates that uptake of phenoxy acid occurs through inhalation and dermal absorption (Kolmodin-Hedman and Erne, 1980). Another study, indicated that dermal absorption was the major route of entry for 2,4,5-T during ground spraying based on the 2,4,5-T residues excreted in the urine (Ferry *et al*., 1982). For workers spraying 2,4,5-T herbicides from containers worn on the back, the average dermal absorption was determined to be 0.08 to 1.85 mg/kg bw (DFG, 1982). Only about 0.002 mg/kg bw was absorbed *via* inhalation.

The exposure of council and forestry workers to 2,4,5-T was monitored for a period of 2 years. It was found that the excessive urine levels of 2,4,5-T fell dramatically once the exposure was minimized through the use of suitable protective clothing. Therefore, the use of protective clothing and adoption of measures to prevent inhalation exposure were deemed essential (Simpson *et al*., 1978).

B24-4.5.2 Distribution

After a large oral dose (25 mg/kg) of the propylene glycol butyl ether ester of 2,4,5-T, sheep showed a peak concentration of 10 mg/L of the unmetabolized ester in the blood four hours after administration (Clark and Palmer, 1971). Oral exposure to 2,4,5-T has also resulted in the presence of 2,4,5-T in the blood plasma of humans (IARC, 1977). 2,4,5-T is widely distributed throughout the body after oral administration of radio-labeled 2,4,5-T to pregnant rats as radioactivity was detected in all of the tissues examined as well as milk and fetuses. Sheep fed

2,4,5,-T also showed the presence of 2,4,5-trichlorophenol, a metabolite of 2,4,5,-T, in muscle, fat, liver, and kidney tissue (Menzie, 1980).

B24-4.5.3 Metabolism

After oral administration of 2,4,5-T to seven human volunteers 88.5% of the 2,4,5-T dose was excreted unchanged (Gehring *et al*., 1973a). Humans do not appear to metabolize 2,4,5-T (Gehring *et al*., 1973a,b).

Sheep receiving large oral doses of 2,4,5-T excreted the compound in the urine mainly unchanged, however, there was a small conversion to 2,4,5-trichlorophenol (Clark *et al*., 1975). Leng (1977) reported that ingestion of 2,4,5-T resulted in excretion of the compound in the urine largely as unchanged phenoxy acid. At higher doses, some conjugation occurred; however, the basic chemical structure of 2,4,5-T was not readily altered within laboratory animals. A similar study with rats found 15 to 30% of a 2,4,5-T dose was excreted as glycine and taurine conjugates and 2,4,5-trichlorophenol (IARC, 1977). The two conjugates were excreted in nearly equal quantities; however, another study with mice showed higher concentrations of the taurine conjugate in the urine (IARC, 1977).

B24-4.5.4 Elimination and Excretion

After seven human volunteers received an oral dose of 2,4,5-T excretion occurred mainly through the urine within 96 hours. A small amount (<1%) of the dose was excreted through the feces during a 48 hour voiding period (Gehring *et al*., 1973a). St. John *et al*. (1964) showed that cattle fed 2,4,5-T eliminated the compound as soluble salts in the urine 2 days after dosing. In addition, sheep that received large oral doses of 2,4,5-T in their feed for 28 days eliminated the compound in the urine, mainly unchanged (Clark *et al*., 1975; Leng, 1977).

Although 2,4,5-T is rapidly eliminated by the body through renal transport most of the time, there are certain circumstances in which large amounts may persist in the plasma and accumulate in the kidneys for long periods of time (Koschier *et al*., 1979). 2,4,5-T binds extensively to the plasma protein which could limit renal clearance. 2,4,5-T has also been found to bind to renal cortex microsomal and cytosol fractions (Koschier *et al*., 1979).

B24-4.6 Exposure Limits

Table B24-12 Existing RfD Values for Exposures to 2,4,5-T

Reference Dose (mg/kg/day)	TCDD Contamination (mg/kg)	Reference	Endpoint	Study	Reference	NOEL (mg/kg/day)	Uncertainty Factor	Study Classification
Acute/Short-term (1-7 days)								
No information found	--	--	--	--	--	--	--	--
Intermediate-term (7 days- Several months)								
No information found	--	--	--	--	--	--	--	--
Long-term (6 months to lifetime)								
0.01	NR	Minnesota State, 2004	Developmental effects (hematologic systems)	--	--	--	--	--
0.01	NR	U.S. EPA, 1989	Increased urinary coproporphyrins	2 year rat feeding study	Kociba *et al*., 1979	3	300	Adequate; Medium confidence in the RfD
0.03[a]	$\leq$ 0.01	WHO, 1981	--	Developmental rat study	Smith *et al*., 1981	3	100	--

[a] Acceptable Daily Intake (ADI) - The amount of a chemical a person can be exposed to on a daily basis over an extended period of time (usually a lifetime) without suffering deleterious effects.

NR Not reported.

The U.S. EPA (1989) established a chronic oral RfD of 0.01 mg/kg/day for 2,4,5-T based upon a NOAEL of 3 mg/kg/day for increased urinary coproporphyrins during a 2 year feeding study in rats (Kociba *et al*., 1979). This study is supported by a three generation rat feed studying with a NOAEL of 3 mg/kd/day for reduced neonatal survival (Smith *et al*., 1981). An uncertainty factor of 300 was applied due to interspecies extrapolation (10), human variability (10) and deficiencies in the chronic toxicity database (3). WHO (1981) utilized the study conducted by Smith *et al*. (1981) to derive an RfD of 0.03 mg/kg/day using an uncertainty factor of 100. Minnesota state published a chronic RfD of 0.01 mg/kg/day derived upon an unknown basis. A chronic RfD value of 0.01 mg/kg/day was selected for risk assessment purposes in this report (U.S. EPA, 1989).

B24-5.0 ENVIRONMENTAL FATE AND EXPOSURE

B24-5.1 Air

B24-5.1.1 Transport and Partitioning

If released into the atmosphere during spray application, 2,4,5-T may exist in a vapour form as fine droplets or adsorbed onto particulates as a result of vapour phase adsorption or wind erosion of treated soils (Norris, 1981). Air borne 2,4,5-T may be physically removed from the atmosphere by settling or washing out in rainfall.

B24-5.1.2 Transformation and Degradation

2,4,5-T has the potential to undergo direct photolysis, due to UV absorption at greater than 290 nm, or to react with photochemically generated hydroxyl radicals.

Table B24-13 Half-life of 2,4,5-T in Air

Conditions	Half-life	Reference
Vapour phase	1.12 day	Norris, 1981
	1.2-4 d; mean=2.3 day	MacKay *et al*., 1997

B24-5.2 Water

B24-5.2.1 Transport and Partitioning

2,4,5-T is not very soluble (Table 24-14), therefore, only small amounts will enter surface water. Stream contamination from spray projects on range and forest lands in Oregon showed that peak 2,4,5-T concentrations seldom exceeded 0.1 mg/L (Norris *et al*., 1971). Residues of 2,4,5-T only persisted for a few hours after application in nearly all of the streams examined (Norris *et al*., 1971).

Based on the pKa value of 2,4,5-T is will be dissociated in water.

B24-5.2.2 Transformation and Degradation

2,4,5-T is not persistent in an aqueous environment as it has a tendency to absorb to clay or biota within a few days (FAO, 2004). In addition, photochemical decomposition and biodegradation are dominant removal mechanisms. Volatilization from water should be minimal as indicated by the Henry's Law constant (Table 24-14) of 2,4,5-T.

Photochemical decomposition of 2,4,5-T occurs in the wavelength region of 300 to 450 nm (Crosby and Wong, 1973). Breakdown of 2,4,5-T to 2,4,5-trichlorophenol occurred slowly with only 10% of the 2,4,5-T undergoing photolysis after 180 hours of irradiation. Photolysis in sunlight is expected to occur at an even slower rate (Crosby and Wong, 1973). Humic substances can photosensitize 2,4,5,-T. Humic induced photoreactions may dominate photodegradation processes when humic substance concentrations exceed 15 mg of organic carbon per liter (Que-Hee *et al*., 1981). The primary products of photodegradation are 2,4,5-trichlorophenol and 2-hydroxy-4,5-dichlorophenoxyacetic acid.

Aerobic degradation of 2,4,5-T results in the formation of 2,4,5-trichlorophenol and 3,5-chichlorocatechol which may further degrade to 4-chlorocatechol or cis,cis-2,4-dichloromuconic acid, 2-chloro-4-carboxy-methylene-but-2-enolide, chlorosuccinic acid and succinic acid (Byast and Hance, 1975). Degradation under anaerobic conditions occur much slower (Sattar and Paasivirta, 1980) and lead to the formation of di- and mono-chlorophenols and 2,5-dichlorophenoxyacetic acid (Mikesall and Boyd, 1985 and Suflita *et al*., 1984).

Table B24-14 Half-life of 2,4,5-T in water

Conditions	Half-life	Reference
	12-42 d; mean= 23 day	Mackay *et al*., 1997
Near-surface; direct photolysis; summer latitude at 40°C	15 day	Que-Hee *et al*., 1981

B24-5.3 Sediment and Soil

B24-5.3.1 Transport and Partitioning

2,4,5-T is moderately mobile in sandy and clay soils (FAO, 2004). Furthermore, 2,4,5-T is expected to vary in its mobility from highly mobile in sandy soil and moderately mobile in clay and silt loams to slightly mobile in mulch (Edwards and Glass, 1971). Groundwater contamination is only expected to occur through large channels and deep soil cracks due to its limited mobility in soil (Edwards and Glass, 1971). 2,4,5-T has a soil adsorption coefficient of 48.63 (JW, 2006).

B24-5.3.2 Transformation and Degradation

2,4,5-T may be degraded chemically or biologically, absorbed in the soil, or leached beyond the depth of plant roots (FAO, 2004). The persistence of 2,4,5-T in soil varies, but normally does not persist from one growing season to the next (WHO, 1979). 2,4,5-T could degrade in one month or may require up to 9 months depending on the climate and soil micro-organism populations (Newman *et al.*, 1952; Warren, 1954; Alexander and Aleem, 1961). Degradation

under anaerobic conditions occurs much slower than under aerobic conditions, therefore, 2,4,5-T persists longer in flooded soils (McCall *et al*., 1981).

2,4,5-Trichlorophenol and 2,4,5-trichloroanisole are the primary degradation products of 2,4,5-T in soil (McCall *et al*., 1981). The anisole is formed through microbial methylation of the phenol. Other metabolites such as 4,6-dichloroescorcinol and 4-chlororesorcinol are also formed from photodecomposition of 2,4,5-T (NRC, 1977). 2,4,5-Trichlorophenol is metabolized in the environment much more readily than the parent compound (Alexander and Aleem, 1961).

The low vapour pressure of 2,4,5-T indicates that volatilization from dry soil surfaces should be insignificant. Volatilization from wet soil surfaces should be minimal as indicated by the Henry's Law constant (Table 24-14) for 2,4,5-T.

Table B24-15 Half-life of 2,4,5-T in Soil

Conditions	Half-life	Reference
	<7 day[a]	JW, 2005
	12-42 d; mean= 23 day	Mackay *et al*., 1997
	21-24 day	FAO, 2004
Favourable conditions	1 month	Warren, 1954
	9 months	Warren, 1954
Flooded soils	≤ 48 months	McCall *et al*., 1981

[a] Soil degradation of 2,4,5-T is considered to be similar to 2,4-D by JW (2006). Therefore, the half-life presented is that of 2,4,-D.

B24-5.4 Other Environmental Media

B24-5.4.1 Transport and Partitioning

The bioconcentration factor (BCF) of 2,4,5-T in static ecosystem tests for fish is 23 to 25 (Kenega and Goring, 1980). In flow through conditions the estimated BCF for fish is 43 (Garten and Trabalka, 1983). JW (2006) reports a BCF of 3.162 for 2,4,5-T. Based on the Log K_{ow} and BCF values for 2,4,5-T significant bioaccumulation within aquatic organisms is not expected to occur.

B24-5.4.2 Transformation and Degradation

Refer to Section B24-4.5 Toxicokinetics.

B24-6.0 SUMMARY

2,4,5-T is in the phenoxyacetic acid family of post-emergent herbicides. It is selective and acts as a plant growth regulator. As an herbicide, 2,4,5-T can be used in crop production, forests, ranges, and aquatic sites. Furthermore, 2,4,5-T can also be used to manage industrial and urban areas. Between 1965 and 1993, approximately 140,000 kg of 2,4,5-T was applied on 20,000 ha of land at CFB Gagetown (JW, 2006). Due to significant 2,3,7,8-TCDD contamination, 2,4,5-T is no longer registered for use in Canada.

Irritation to the eyes and the respiratory tract will occur when humans are exposed to 2,4,5-T on a short-term basis (NIOSH, 2005). Long term or repeated exposures of 2,4,5-T to humans may cause adverse reproductive/developmental effects (NIOSH, 2005). Most of the 2,4,5-T toxicity studies performed in rodents showed adverse systemic effects. There were also signs of developmental impairment when the animals were repeatedly exposed 2,4,5-T. Maternal toxicity, based on systemic effects and behavioral signs were also observed. Furthermore, chronic feeding studies in rodents have shown related increases in the incidence of cancers when exposed to 2,4,5-T. Chlorophenoxy herbicides have been classified as possible human carcinogens (Group 2B) by IARC (1987). This classification was based on limited evidence for carcinogenicity of chlorophenoxy herbicides in humans and inadequate evidence for 2,4,5-T carcinogenicity in animals. All of the animal carcinogenicity studies were considered inadequate due to the small numbers of animals used (IARC, 1987).

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