B18-1.0 DICHLORPROP (2,4-DP)

B18-1.1 Background Information

IUPAC: 2-(2,4-dichlorophenoxy)propanoic acid

CAS: (±) 2-(2, 4-dichlorophenoxy) propionic acid.

CASRN: 53404-31-2

DICHLORPROP USAGE:

2,4-DP, and its salts and esters could be used alone or in combinations with other herbicides for the purpose of broad leaf weed control. 2,4-DP, was used in agriculture, landscaping , and forestry industries (CEPA, 2001).

Dichlorprop was the active ingredient of several herbicide products that were applied at the CFB Gagetown. These include, LV Brush Killer 700® which was applied in 1998 in conjunction with Dycleer® (Dicamba). Silvaprop® was applied in 1984. Furthermore, Silvaprop® was applied in conjunction with herbicide product Dycleer LH® (Dicamba) in 1985.

Dichlorprop was used at CFB Gagetown between 1984 and 1988 (JW, 2006).

Table B18-1 2,4-DP Usage at CFB Gagetown[a]

Year	2,4-DP Applied (kg)	Total Area Treated (ha)
1984	9156.9	2990
1985	133.56	53
1988	6581.8	1270.23
Total	**1.6E+04**	**4.3E+03**

[a] Adapted from JW, 2006.

B18-2.0 CHEMICAL AND PHYSICAL PROPERTIES

Formula: $C_9H_8Cl_2O_3$

Activity: Phenoxy acid herbicide, with similar activity as the auxins. 2,4-DP has two optical isomers. The (+) isomer is biologically active as an herbicide (CEPA, 2001).

Notes: 2,4-DP is often referred to as 2,4-DP for short form. 2,4-DP belongs to the family of phenoxy acid herbicides. Both isomers of 2,4-DP could be found in commercial preparations as a racemic mixture. Preparations of the (+) isomer alone could also be found commercially (CEPA, 2001). However 2,4-DP based herbicides are no longer registered for use in Canada, and the United States (Orme *et al*., 2006).

Structure:

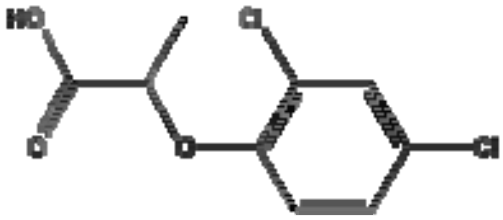

Figure B18-1 2,4-DP CASRN: 120-36-5 Structure

Table B18-2 Chemical and Physical Properties of 2,4-DP

Chemical/Physical Property	Result	Reference
Colour/Form	Colourless, crystalline solid	CEPA, 2001
Odour	Odourless	CEPA, 2001
Dissociation Constant (pKa)	2.855 - 3.1	Mackay *et al*., 1997; JW, 2006
Henry's Law constant	2.69 x 10^{-4} Pa m^3/mol	Mackay *et al*., 1997
	8.68 x 10^{-11}atm-m^3/mole (25ºC)	JW, 2006.
Log K_{ow}	1.77 - 3.43	Mackay *et al*., 1997
	4.52	U.S. EPA, 2000
Melting Point	116-118.1ºC	Mackay *et al*., 1997
Molecular Weight	235	CEPA, 2001
Vapour Pressure	<1.0x10^{-5} - 4.50x10^{-4} P	Mackay *et al*., 1997
	7.50 x 10^{-8} mm Hg (20ºC)	JW, 2006
Water Solubility	350 mg/L (20ºC)	Mackay *et al*., 1997; JW, 2006

B18-3.0 PMRA EVALUATION

No information found.

B18-4.0 TOXICOLOGICAL SUMMARY

The following toxicological profile for 2,4-DP is a short summary based on the California Environmental Protection Agency's draft report entitled "Evidence on the developmental and

reproductive toxicity of 2,4-DP" and WHO's Guidelines for Drinking-water Quality. Only the toxicological studies appropriate for guideline development were highlighted below.

B18-4.1 Human Health Effects

Table B18-3 Human Health Effects Resulting from Acute Exposure to 2,4-DP Containing Herbicides[a, b]

Exposure	Effects	Response
Acute	Vital signs	Miosis, coma, fever, hypotension, emesis, tachycardia, bradycardia, ECG abnormalities, muscle rigidity, possible respiratory failure, pulmonary edema, and rhabdomyolysis may occur. Deaths have resulted from cardiorespiratory arrest.
	HENNT	Eye, nose, and mouth irritation are possible with direct contact.
	Cardiovascular	Tachycardia, bradycardia, ECG abnormalities, asystole, other dysrhythmias, and hypotension have been reported with overdose. Deaths have resulted from cardiorespiratory arrest.
	Respiratory	Ingestion of large amounts may cause bradypnea, respiratory failure, hyperventilation, or pulmonary edema.
	Neurologic	Vertigo, headache, malaise, and paresthesias may occur. Muscle twitching, spasms, profound weakness, polyneuritis, and unconsciousness may occur. Peripheral neuropathies
	Gastrointestinal	Nausea, vomiting, and diarrhea have been reported. Necrosis of the gastrointestinal mucosa has been reported.
	Hepatic	Elevated LDH, AST (SGOT), and ALT (SGPT) have been reported.
	Genitourinary	Albuminuria and porphyria may occur; renal failure due to rhabdomyolysis is also possible.
	Fluid-electrolyte	Hypocalcemia, hyperkalemia, and hypophosphatemia.
	Hematologic	Thrombocytopenia is the primary hematologic effect. Leukopenia has also been reported.
	Dermalogic	Direct contact may cause skin irritation.
	Musculoskeletal	Muscle cramps, muscle rigidity, elevated creatinine kinase, and rhabdomyolysis may occur. EMG abnormalities.
	Endocrine	Hypoglycemia has been reported.

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

B18-4.2 Health Effects by Route of Exposure

B18-4.2.1 Oral Exposure

Table B18-4 Mammalian LD_{50} Values Resulting from Oral Exposure to 2,4-DP

Test Type	Test Organism (Species/Sex)	LD_{50} (mg/kg)	Reference
Acute	Mice	300 mg/kg	RTECS, 1978
	Mice	400 mg/kg	Meister *et al*., 1984
	Rat	800 mg/kg	Meister *et al*., 1984

Table B18-5 Mammalian Effects Resulting from Oral Exposure to 2,4-DP

Test Organism (Species)	Exposure	Dose (Duration)	Response	Reference
Sub-chronic				
Mice	Diet	2,700 ppm[a] for 3 weeks	Increased liver weight, circulating alkaline phosphatase, and alanine aminotransferase. Central lobular hypertrophy with retarded body weight gain and eosinophilia.	CEPA, 2001
Rat	Diet	150 mg/kg/day for 2 weeks	Induction of rat liver peroxisomal enzymes, hepatomegaly, decreased serum cholesterol and triglycerides.	Koibuchi *et al*., 1993
Mice	Diet	3600 ppm[a] for 3 months	Disrupted lipid metabolism with signs of anemia.	CEPA, 2001
Chronic				
Fischer 344 rats	Diet	0, 100, 300, 1,000, 3,000 mg/kg/day for two years	At the 3,,000 mg/kg/day dose level, survival was slightly reduced in females. Body weight was depressed by 10% in both males and females; diffuse hepatocellular swelling and deposition of brown pigment in liver cells; mild anaemia, decreased haematocrit, erythrocyte count, and haemoglobin. Pigmentation increased in kidneys at 1,000-3,000 mg/kg/day dose level, with slight degeneration of the tubular epithelium. Specific gravity of protein was decreased as well.	IET, 1984b
Rats	Diet	94 mg/kg/day 2 for 5 months.	Body weight loss, increased liver weight, urinary obstruction, leg paralysis. Systemic cholesterol concentrations were reduced by 50%.	Ohta *et al*., 1987
Wistar rats	Diet	2,000 ppm for 2 generations	Decreased cholesterol and triglycerides, increased alkaline phosphatase, increased urinary crystals, and decreased hematocrit.	Hellwig, 1992

[a] (+) isomer of 2,4-DP used.

Table B18-6 Mammalian Developmental Effects Resulting from Oral Exposure to 2,4-DP

Test Organism (Species)	Exposure	Dose (Duration)	Response	Reference
Mice	Gavage	300-500 mg/kg/day	Decreased fetal weight, increased incidence of fused ribs, resorptions, postimplant loss, and cleft palate.	Roll and Matthiaschk, 1983
Mice	Gavage	300-500 mg/kg/day[a]	Decreased fetal weight, increased incidence of malformed vertebrae, early resorptions, post implantation loss, cleft palate, fused ribs, and exencephaly.	Roll and Matthiaschk, 1983
Sprague Dawley rats	Gavage	300 mg/kg/day	Developmental effects: Decreased litter weight. Maternal effects: decreased food intake and weight gain.	Huntingdon, 1978

Table B18-6 Mammalian Developmental Effects Resulting from Oral Exposure to 2,4-DP

Test Organism (Species)	Exposure	Dose (Duration)	Response	Reference
Wistar rats	Gavage	226 mg/kg/day	Developmental effects: decreased birth-weight, decreased litter size, increased incidence of still births, and increased incidence of dilated renal pelvis. Maternal effects: Decreased food intake and weight gain.	Hellwig, 1992
Wistar rats	Gavage	160 mg/kg/day[a]	Developmental effects: increased incidence of extra ribs, decreased fetal weight, decreased bone ossification, increased hydro ureter. Maternal effects: Decreased food intake, and weight gain.	Hellwig, 1993b
F344 rats	Diet	15, 50 for 13 mg/kg/day weeks	Decrease absolute and relative testes weight.	Inst Environ Toxicology, 1985
F344 rats	Diet	11-116 mg/kg for 24 months	Decreased absolute ovary weight in 26 weeks, at 11 mg/kg/day. Decreased absolute and relative ovary weight in 78 weeks, at 11 mg/kg/day. Increased absolute and relative ovary weight in 104 weeks, at 4 mg/kg/day. Increased absolute testes weight at 26 and 52 weeks, at 116 mg/kg. Decreased absolute and relative testes weight at 78 weeks.	IET, 1984b
Wistar rats	Diet	11, 53 mg/kg/day for 4 weeks	Increased absolute testes weights in males	Kirsch *et al*., 1986
Dutch-belted rabbits	Gavage	75 mg/kg/day	Developmental effects: Decreased fetal weight, 3 incidences of malformed fetuses. 4 maternal mortalities were observed.	Hazleton, 1979
Himalayan Rabbits	Gavage	100 mg/kg/day[a]	Developmental effects: Decreased fetal weight, increased incidence of extra ribs. Maternal effects: Decreased food intake.	Hellwig, 1993a

[a] (+) isomer of 2,4-DP used

Table B18-7 Mammalian Reproductive Effects Resulting from Oral Exposure to 2,4-DP

Test Organism (Species)	Exposure	Dose (Duration)	Response	Reference
Beagle dogs	Diet	48 mg/kg/day for 13 weeks	Testicular effects with high mortality	Reuzel *et al*., 1980
Beagle Dogs	Diet	4, 24 mg/kg/day for 12 months	Increased relative testes weight, observed at dose 4 mg/kg. Testicular pathology with high mortality at dose 24 mg/kg/day	Bachmann, 1997

B18-4.2.1.1 No Observed Adverse Effect Levels

Table B18-8 Mammalian NOAELs and LOAELs for Oral Exposure to 2,4-DP

Test Organism (Species)	Effect	Value (mg/kg/day)	Endpoint	Reference
Sub-chronic				
Rats	NOEL	12.4	Systemic toxicity	WSSA, 1985
Chronic				
Fischer 344 rats	NOAEL	3.64 (M) 13.1(F)	Renal toxicity	IET, 1984b

B18-4.2.2 Dermal Exposure

Table B18-9 Mammalian Acute LD_{50} Value Resulting from Dermal Exposure to 2,4-DP

Test Type	Test Organism (Species/Sex)	LD_{50} (mg/kg)	Reference
Acute	Mice	1,400	WSSA, 1985

B18-4.2.3 Inhalation Exposure

No data found.

B18-4.3 Carcinogenicity

2,4-DP belongs to the chlorophenoxy class of herbicides. It has been recognized and classified as a possible carcinogen (Group 2B) to humans, by the International Agency for Research on Cancer (IARC). IARC came to this conclusion on the basis of epidemiological studies that showed associations between exposures of 2,4-DP in soft tissues with sarcoma and non-Hodgkin's lymphoma that resulted (IARC, 1987).

Table B18-10 Animal Carcinogenicity Data

Test Subjects	Exposure	Dose (Duration)	Response	Reference
CD-1 Mice	Diet	0, 25, 100, 300 mg/kg/day for 18 months	Increased incidence of benign hepatomas[a]	CDC, 1986
Mice	Oral	Unspecified	High incidence of palpable abdominal masses.	CDC, 1979
Rats	Oral	25, 50, 200 mg/kg/day for >15 months	No increase in the incidence of carcinogenesis.	CDC, 1980

[a] Authors speculate that the cause was increased metabolic burden on the liver, which impaired metabolic process needed for the suppression of carcinogenesis. Authors concluded dichlorprop was not carcinogenic at the doses tested.

A number of mutagenicity tests have been conducted on 2,4-DP. These include Ames tests, Chinese hamster ovary HPGRT locus assays, and reverse mutation assays. Similarly chromosome abnormality tests such as metaphase analysis of human lymphocytes, chromosome

aberration assays in human lymphocytes, sister chromatid exchange, bone marrow chromosome aberration test in Chinese hamster cells were conducted. DNA damage, and *in vivo* micronucleus tests were also conducted for both 2,4-DP, 2,4-DP(+), their salts and esters. No mutagenic effects were recorded but possible effects on chromosomes were seen in sister chromatid exchange, hamster bone marrow, and human lymphocyte studies (CEPA, 2001).

B18-4.4 Populations at Special Risk

No information found.

B18-4.5 Toxicokinetics

Phenoxy acid herbicides are usually completely absorbed from the GI tract from rats but minimally through dermal administration. Furthermore, Phenyxy acid herbicides are highly protein bound, and tend to have high volume of distribution. 2,4-DP is of no exception. Highest concentrations of 2,4-DP will be found in lungs, liver, kidneys, thyroid and adrenal glands, between 1 to 3 hours after its initial administration (CEPA, 2001).

2,4-DP is excreted largely unchanged (>70% in male rats, >81% in female rats) by the kidney's organic acid transport (OCT) mechanism. It was observed that ascorbic acid excretion was more than doubled, indicating that 2,4-DP is not only excreted by OTCs but will also upregulate OTC's expression (CEPA, 2001).

Prior to excretion, a small amount of 2,4-DP may be hydrolysed and/or conjugated, and at high doses, enterohepatic circulation of 2,4-DP will occur *via* biliary excretion (CEPA, 2001).

When 2,4-DP was orally administered to Wistar rats, it was found that 2,4-DP's concentrations in bile was twice as high as in serum after 8 hours. Serum half-life was evaluated to be between 9.5 to 10.5 hours for a dose ranging from 30 to 300 mg/kg. Furthermore, it was found that the leucine conjugate of 2,4-D, had a higher bilary concentration but shorter half life than unconjugated 2,4-DP (CEPA, 2001).

2,4-DP does not only upregulate OTCs, it will also induce such enzymes as CYP4D1, as well as aniline hydroxylase, morphine N-demethylase, cytochrome bs. Conversely, *in vitro* studies, it was found that 2,4-DP will bind and inhibit hepatic glutathione metabolism (CEPA, 2001).

B18-4.6 Exposure Limits

Table B18-11 Existing RfD Values for 2,4-DP Exposures

Reference Dose (mg/kg/day)	Route of Exposure	Reference	Endpoint	Study	References	NOEL (mg/kg/day)	Uncertainty Factor
Acute/Short-term (1-7 days)							
0.5[b]	Oral	EFSA, 2005	--	Rabbit teratogenicity study	--	50	100
Intermediate-term (7 days- Several months)							
0.35[c]	Oral	PSD, 2007	Kidney, liver, and blood effects	Rat 90 day study	--	35	100
Long-term (6 months to lifetime)							
0.0364[a]	Oral	WHO, 1996	Renal toxicity	2 year study in rats	IET, 1984a	3.64	100
0.06[b]	Oral	EFSA, 2005	Chronic nephropathy	18 month dietary study in mice	--	6.0	100

[a] Tolerable daily intake reported. Regulatory value equivalent to the acceptable daily intake established by the European Commission Scientific Committee on Food. TDI is normally used for food contaminants.

[b] Reference dose values based on dichlorprop-p which is the R-isomer of 2,4-DP.

[c] Acceptable Operator Exposure limit (AOEL).

For the purposes of this report, acute/short-term oral RfD of 0.5 mg/kg/day (EFSA, 2005), intermediate-term oral RfD of 0.35 mg/kg/day (PSD, 2007), and long-term RfD of 0.06 mg/kg/day (EFSA, 2005) were selected.

B18-5.0 ENVIRONMENTAL FATE AND EXPOSURE

2,4-DP has low vapour pressure (Table B18-12) and thus volatilization would only occur to a minor extent. Furthermore, 2,4-DP also has a low Henry's Law constant of 8.68×10^{-11} atm-m^3/mole (JW, 2006). This indicates that it would have little tendency to escape from an aqueous solution. Hence, 2,4-DP will not be expected to be found in air with the exception as a consequence of direct aerial application of compounds to agricultural and non agricultural areas. Since 2,4-DP is a chlorophenoxy herbicide, it is considered to have only marginal potential for leaching to groundwater (U.S. EPA. 1985). In waters with higher pH, phenoxy herbicide esters are usually hydrolysed to the anionic forms. Whereas in waters with lower pH, photodegradation dominate the degradation process (WHO, 1996). Half-life of 2,4-DP in groundwater is estimated between 196 days to over 1,200 days (JW, 2005). 2,4-DP does not adsorb to soil, and will have a half life roughly of 10 days (JW, 2006) with a range between 10 to 12 days (WHO, 1996). In soil 2,4-DP will degrade to 2,4-dichlorophenol, and will disappear completely in 14 days. Biodegradation is the primary route of elimination for chlorophenoxy herbicides from the environment. Photolysis and hydrolysis also contribute to their removal. 2,4-DP is estimated to have a bioconcentration factor of 3.162 (JW, 2006).

Table B18-12 Half-life of 2,4-DP in the Environment

Conditions	Environmental Media	Half-life	Reference
Groundwater	Water	196-1,200 days	JW, 2005
Microbial degradation	Soil	10 days	JW, 2005

B18-6.0 SUMMARY

Dichlorprop and its salts and esters can be used alone or in combinations with other herbicides for the purpose of broad leaf weed control (CEPA, 2001). As a systemic herbicide, dichlorprop was used in agriculture, landscaping, and forestry industries (CEPA, 2001). As a member of the phenoxy acid family of herbicides, dichlorprop is often referred to as 2,4-DP. While, 2,4-DP has similar activities to the auxins, it is made up of two optical isomers. Both isomers of 2,4-DP can be found in commercial preparations as a racemic mixture. However, only the (+) isomer is biologically active as an herbicide (CEPA, 2001). Preparations of the (+) isomer alone can also be found commercially (CEPA, 2001). Between 1984 and 1988, approximately, 16,000 kg of dichlorprop was applied over an area of 4,300 ha at CFB Gagetown (JW, 2006). 2,4-DP is persistent and mobile in soils, hence it could pose as a problem to groundwater supplies. 2,4-DP is no longer registered for use in Canada and the United States.

2,4-DP has low acute toxicity through the oral and dermal routes of exposure. However, 2,4-DP will produce adverse developmental/reproductive effects in rodents. IARC (1987) classified 2,4-DP as a possible human carcinogen (Group 2B).

B18-7.0 REFERENCES

Bachmann, S., Deckardt, K., Gembardt, C. and Hildebrand, B. 1997. Dichlorprop-P – Chronic oral toxicity study in beagle dogs - administration in the diet for 12 months. BASF Akteingesellschaft. 33D0187/91167. Cited In: CEPA, 2001.

CDC Research Laboratories. 1979. Oncogenicity study in mice with 2,4-DP acid. CDC Research Laboratories, Inc. CDC-AM-002-77. Cited In: CEPA, 2001.

CDC Research Laboratories. 1980. Oncogenicity study in rats with 2,4-DP acid. CDC Research Laboratories, Inc. CDC-AM-001-77. Cited In: CEPA, 2001.

CDC Research Incorporates. 1986. Oncogenicity study in mice with 2,4-DP acid. (unpublished study no. CDC-AM-002-77, submitted to Union Carbide, Agricultural Products Company, Research and Development Center). Cited In: WHO, 1996.

CEPA. 2001. Evidence on the developmental and reproductive toxicity of 2,4-DP. Reproductive and cancer hazard assessment section. Office of environmental health hazard assessment. California environmental protection agency, October, 2001.

EFSA. 2005. Conclusion of the Peer Review of Dichlorprop-p. European Food Safety Authority Scientifc Report 52: 1-67. Available at: http://www.efsa.europa.eu/etc/medialib/efsa/science/praper/conclusions/1434.Par.0001.File.dat/praper_concl_sr52_dichlorprop-p_en1.pdf. [September 27, 2006].

Hazleton. 1979. 2,4-DP: Oral teratogenicity study in the Dutch-belted rabbit. Hazleton Laboratories Europe, Ltd. 1577R-277/5. Cited In: CEPA, 2001.

Hellwig, J. 1992. Reproduction study with 2,4-DP in rats: continuous dietary administration over 2 generations (2 litters in the first and 2 litters in the second generation). BASF AG. Sponsor Report # 89/0474. Cited In: CEPA, 2001.

Hellwig, J.1993a. Prenatal toxicity of dichlorprop-P in rabbits after oral administration (gavage). BASF AG. Sponsor Report # 93/10230. Cited In: CEPA, 2001.

Hellwig J. 1993b. Prenatal toxicity of dichlorprop-P in rats after oral administration (gavage). BASF AG. Sponsor Report # 93/10227. Cited In: CEPA, 2001.

HSDB. Hazardous Substances Data Bank. 2003. Dichlorprop. U.S. National Library of Medicine's Toxicology Data Network (TOXNET). Available at: http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB. [August 22, 2006].

Huntingdon Research Center. 1978. Three generation study of 2,4DP technical acid in rats. Cited In: CEPA, 2001.

IARC. 1987. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans. Vol 41. Some Halogenated Hydrocarbons and Pesticide Exposures. Supplement 7. International Agency for Research on Cancer, World Health Organization. Cited In: CEPA, 2001.

IET. 1984a. 2,4-DP acid 24-month oral chronic dietary study in rats. Union Carbide.(unpublished report). Institute of Environmental Toxicology . Cited In: WHO, 1996.

IET. 1984b. 2,4-DP Acid (2-(2,4-dichlorophenoxy)propanoic acid): 24-month oral chronic dietary study in rats. Institute of Environmental Toxicology (Tokyo). 85/071. Cited In: CEPA, 2001.

IET. 1985. A365 (2,4-DP acid): 24-month oral chronic dietary study in rats (13 week supplemental study). The Institute of Environmental Toxicology (Tokyo). Cited In: CEPA, 2001.

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.

Kirsch, P., Deckart, K., Gembardt, C., Hasenohr, K., Kuhborth, B. and Hildebrand, B.1986. Report on the comparative study of the toxicity of the racemate and D-form of 2,4-DP in rats after four week administration in the diet. BASF AG. Project 30S0048. Cited In: CEPA, 2001.

Koibuchi, Y., Yamada, J., Watanabe, T. and Suga, T. 1993. Lack of stereoselectivity in rat liver peroxisomal enzyme induction by optically active phenoxyacetic acids. Biol Pharm Bull16: 39-42. Cited In: CEPA, 2001.

Mackay, D., Shiu, W.Y., and Ma, D.C. Year. 1997. Illustrated handbook of Physical-Chemical Properties and Environmental Fate for Organic Chemicals. Volume V, Pesticide Chemicals. Lewis Publishers, Boca Raton. New York.

MEDITEXT®. 2006. TOMES® Information System Micromedex, Inc., Englewood, Co; CCIS Volume 129, expires Aug, 2006. Cited In: HSDB, 2003.

Meister, R.T., Berg, G.L., Sine, C., Meister, S. and Poplyk, J. eds. 1984. Farm Chemicals Handbook, 70th ed. Meister Publishing Co., Willoughby, OH.

RTECS. 1978. National Institute of Occupational Safety and Health. Registry of Toxic Effects of Chemical Substances (RTECS). Cincinnati, OH, US Department of Health and Human Services, 1978. Cited In: WHO, 2006.

Ohta, F., Gaudemer, F. and Lutton, C. 1987. Hypocholesterolemic activity of racemic dichlorophenoxypropionic acid or its enantiomers in rats. *Bull Environ Contam Toxicol* 39: 966-73. Cited In: CEPA, 2001.

Orme, S. and Kegley, S. 2006. Dichlorprop. PAN Pesticide Database, Pesticide Registration Status Pesticide Action Network, North America. San Francisco, CA. 2006. http://www.pesticideinfo.org/Detail_ChemReg.jsp?Rec_Id=PC33424

PMEP. 1985. Dichlorprop (Weedone, Cornox) Herbicide Profile 3/85. Pesticide Management Education Program, Cornell University, Ithaca, NY. Available at: http://pmep.cce.cornell.edu/profiles/herb-growthreg/dalapon-ethephon/dichlorprop /herb-prof-dichlorprop.html [August 22, 2006]

PSD. 2007. Toxicological Endpoints Database. U.K. Product Safety Directorate Databases. Available at: https://secure.pesticides.gov.uk/TEAWeb/tea_results.asp [Feb 16, 2007]

Reuzel, P.GF., Immel, H.R. and Feron, V.J. 1980. Sub-chronic (13-week) oral toxicity study with 2,4-dichloro-phenoxy propionic acid in beagle dogs. BASF AG. R6246. Cited In: CEPA, 2001.

Roll, R. and Matthiaschk, G. 1983. Comparative studies on the embryotoxicity of 2-methyl-4-chlorophenoxyacetic acid, mecoprop and dichlorprop in NMRI mice. *Arzneimittelforschung* 33, 1479-83. (translated from the German by Berkeley Scientific Translation Service, Inc.) Cited In: CEPA, 2001.

Rumack, B.H., and Hall, A.H. Eds. 2006. POISINDEX® Information system Micromedex, Inc., Englewood, CO. CCIS Volume 129, expires Aug 2006. Cited In: HSDB, 2003.

U.S. EPA. 1985. EPA draft final list of recommendations for chemicals in the national survey for pesticides in groundwater, August 1985. United States, Environmental Protection Agency. Chemical regulations reporter 9(34): 988. Cited In: WHO, 1996.

U.S. EPA. 2000. Estimation Interface Program (EPI) Suite. Version 3.12 (December 8, 2005). Available at: http://www.epa.gov/opptintr/exposure/pubs/episuite.htm. [February 14, 2007].

WSSA. 1983. Herbicide Handbook Committee. Herbicide handbook of the weed science society of America, 5th ed. Weed Science Society of America, Champaign, IL. 515 pp. Cited In: PMEP, 1985.

WHO. 1996. Chlorophenoxy herbicides (excluding 2,4-D and MCPA) in Drinking-water Background document for development of WHO Guidelines for Drinking-water Quality. World Health Organization, Geneva, Switzerland. Available at: http://www.who.int/ water_sanitation_health/dwq/chemicals/chlorophenoxyherb.pdf [August 23, 2006]