B6-1.0 DICAMBA

B61.1 Background Information

IUPAC: 3, 6-Dichloro-o-Anisic Acid

CAS: 2-methoxy-3,6-dichlorobenzoic acid or 3,6-dichloro-2-methoxy-benzoic acid

CASRN: 1918-00-9

DICAMBA USAGE:

Dicamba is commonly used for the control of annual and perennial broadleaf weeds, brush, and vines in rangeland and non-cropland areas. The non-croplands include fenced rows, roadways, rights-of-way, maintenance of wildlife openings, and non-selective forest brush control (USDA/FS, 1999).

Dicamba was the active ingredient of several herbicide products that was applied at the CFB Gagetown. These include, Dycleer® which was applied in 1989, and was applied in conjunction with LV Brush Killer 700® in 1988 and in conjunction with 2,4-D in years 1989 and 2000. Herbicide product Dycleer 24® was applied in years 1978, 1983 and 1994. Dycleer 10p® was applied in years 1980 and 1983. Furthermore, dicamba was also an active ingredient in the herbicide product Dycleer LH which was applied in years 1984 and 1985.

Dicamba was used at CFB Gagetown between 1978 and 2000 (JW, 2006).

Table B6-1 Dicamba Usage at CFB Gagetown[a]

Year	Amount of Dicamba Applied (kg)	Total Area Treated (ha)
1978	84.2	4.2
1980	6,867	1,566
1983	5,834	1,558
1984	8,970	2,990
1985	165	53
1988	6,677	1,270
1989	1,112	556.0
1994	278.0	138.5
2000	29.8	14.8
Total	**3.0E+04**	**8.2E+03**

[a] Adapted from JW, 2006.

B6-2.0 CHEMICAL AND PHYSICAL PROPERTIES

Formula: $C_8H_6Cl_2O_3$

Activity: Herbicide. Dicamba is an auxin agonist which causes uncontrolled cell division and growth in sensitive plants. Destruction of vascular tissue, as a result of uncontrolled growth of the stems, petioles and leaves, leads to plant death (U.S. EPA, 2006).

Notes: Dicamba is inherently an acid, and it will form salts in aqueous solutions. Different dicamba salts are formulated for herbicidal use and they include dimethylamine salt, sodium salt, isopropylamine salt, diglycolamine salt, and potassium salt. There are approximately 434 active products of dicamba formulated to date (U.S. EPA, 2006).

Structure:

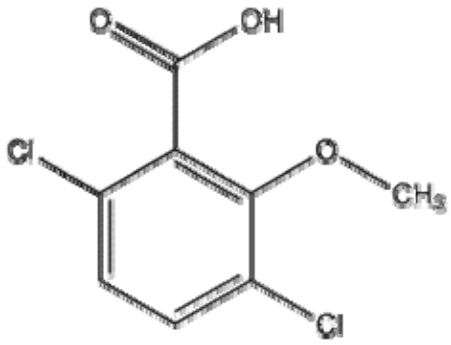

Figure B6-1 Dicamba CASRN: 1918-00-9

Table B6-2 Chemical and Physical Properties of Dicamba

Chemical/Physical Property	Result	Reference
Colour/Form	White crystalline solid, pale buff crystalline solid	Kidd and James, 1991
pH	2.5-3.0	U.S. EPA, 2006
Density, bulk density, or specific gravity (25ºC)	1.57 g/mL	U.S. EPA, 2006
Dissociation Constant (pKa)	1.97	JW, 2006
	1.87-1.95	Hornsby *et al*., 1996; Worthing, 1991; Tomlin, 1994
Henry's Law constant	0.000022 to 0.0918	Mackay *et al*, 1997; Howard, 1991
	2.18×10^{-9}	JW, 2006
Octanol/Water Partition Coefficient, Log K_{ow}	-1.69-3.01	Mackay *et al*, 1997
	2.14	U.S. EPA, 2000
Melting Point	114-116 ºC	Kidd and James, 1991; Mackay *et al*, 1997

Table B6-2 Chemical and Physical Properties of Dicamba

Chemical/Physical Property	Result	Reference
Molecular Weight	221.04	Mackay *et al*, 1997; U.S. EPA, 2006
Odour	Odourless	Kidd *et al*., 1991
Vapour Pressure (25ºC)	0.001 Pa	Montgomery ,1993
	4.00×10^{-5} Pa	Worthing, 1991
	3.38×10^{-5} mm Hg	JW, 2006
	0 Pa	Halfon *et al.*, 1996
	Negligible	Tomlin, 1994
Water Solubility (25ºC)	4500-7900 mg/L	Mackay *et al*, 1997; Kidd *et al*., 1991
	8310 mg/L	JW, 2006

B6-3.0 PMRA EVALUATION

Dicamba is scheduled to be re-evaluated under Program 3 in PMRA's work plan published on December, 2004.

B6-4.0 TOXICOLOGICAL SUMMARY

Most studies conducted to evaluate adverse health effects of dicamba used technical grade dicamba. The dicamba formulations used in Gagetown included other ingredients such as 2,4-D and dichloroprop with other possible contaminants such as dioxins. The U.S. EPA has established a lifetime health advisory level of 200µg/L for dicamba in drinking water (JW, 2006).

B6-4.1 Human Health Effects

Table B6-3 Human Health Effects Resulting From Acute Exposure to Dicamba[a,b]

Exposure	Effects	Response
Acute	Vital Signs	Irritation of the mucous membranes, dyspnea, pulmonary irritation, incontinence, vomiting, anorexia, muscle spasms and weakness, and skin irritation. Eye irritation may occur. Conjunctival swelling and corneal clouding were noted when eyes were exposed to dicamba. Effects generally lasted 5 to 7 days. The dimethylamine salt is especially irritating.
	Respiratory	Respiratory irritation may occur. Dyspnea and cyanosis are signs of dicamba intoxication.
	Neurologic	CNS depression may occur.
	Gastrointestinal	Vomiting, anorexia, and weight loss have been reported with human exposure to the dimethylamine salt.
	Dermatologic	Dermal exposures may result in skin irritation or burns, since dicamba is mildly corrosive.
	Musculoskeletal	Myotonia may occur following severe intoxication. Muscular weakness was reported after human exposure to the dimethylamine salt.

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

B6-4.2 Health Effects by Route of Exposure

B6-4.2.1 Oral Exposure

B6-4.2.1.1 Death

Table B6-4 Mammalian LD_{50} Values Resulting From Oral Exposure to Dicamba

Test Organism (Species/Sex)	LD_{50} (mg/kg)	Reference
Acute		
Mice	,1190	Kidd *et al*., 1991; Stevens *et al*., 1991
Rat	≥2,740 mg/kg	U.S. EPA, 2006
Rats	757-,1707	Kidd *et al*., 1991; Stevens *et al*., 1991
Guinea pigs	566-3,000	Kidd *et al*., 1991; Stevens *et al*., 1991
Rabbits	2,000	Kidd *et al*., 1991; Stevens *et al*., 1991

B6-4.2.1.2 Systemic Effects

Table B6-5 Mammalian Systemic Effects Resulting From Oral Exposure to Dicamba

Test Organism (Species)	Daily Dose (Duration)	Response	Reference
Sub-chronic			
Wistar rats	119 and 364 mg/kg/day for 15 weeks	Increased relative liver/body weight ratios	Edson *et al*., 1965
Sprague Dawley rats (m/f)[A]	413 mg dicamba per kg diet per day for 13 weeks	Necrosis and vacuolization of the liver were reported	NAS, 1977
Male rats	0, 40. 1, 238. 7, 479.4, ,1000 mg/kg/day (unspecified period)	Decreased body weight gains, increased liver weight and increased centrolobular, hepatocyte, hypertrophy and hepatocellular pigmentation.	U.S. EPA, 2006
Female rats	0, 43.2, 266.4, 535.6, 1065.3 mg/kg/day (unspecified period)		
Chronic			
Rats	Male: 0, 2, 11, 107, mg/kg/day (unspecified period)	Carcinogenesis. Study found that dicamba was not carcinogenic.	U.S. EPA, 2006
	Female: 0, 3, 13, 127 mg/kg/day (unspecified period)		
Dogs	0, 2, 11, 52 mg/kg/day (unspecified period)	Unspecified effects.	U.S. EPA, 2006

[a] m/f denotes male and female.

B6-4.2.1.3 Neurological Effects

Table B6-6 Mammalian Neurologic Effects Resulting from Oral Exposure to Dicamba

Test Organism (Species)	Daily Dose (Duration)	Response	Reference
Acute			
Rat	0, 300, 600, 1200 mg/kg	Impaired respiration, rigidity upon handling, prodding, or dropping, impaired gait and righting reflex in both sexes.	U.S. EPA, 2006.
Sub-Chronic			
Rat	Male: 0, 197, 1401.4, 4767.9 mg/kg/day	Rigidity body tone and slightly impaired righting reflex and gait.	U.S. EPA, 2006.
	Female: 0, 253. 4, 472, 1028.9 mg/kg/day		

B6-4.2.1.4 Reproductive/Developmental Effects

Table B6-7 Mammalian Reproductive and Developmental Effects Resulting from Oral Exposure to Dicamba

Test Organism (Species)	Exposure	Dose (Duration)	Response	Reference
Rat	Diet	Male: 0, 40, 122, 419 mg/kg/day	Parental/Systemic: Slowing righting reflex.	U.S. EPA, 2006
			Reproductive: delayed sexual maturation in first generation females	
		Female: 0, 45, 136, 450 mg/kg/day	Offspring: impaired pup growth, decreased pup weights	
Rat (CD)	Oral	0, 0.25, 2.5, 5, 12.5 or 25 mg/kg/day for 3 generations.	There were no effects on fertility, viability or pup development.	Witherup *et al*., 1966
Rat (albino)	Gavage	0, 0.5, 1, 3, 10 or 20 mg/kg/days 6 to 18 of pregnancy.	No teratogenic or foetotoxic effects were noted	Wazeter *et al*., 1977
Rat	Oral	0, 64, 160, 400 mg/kg/day	Maternal: increased mortality rates, decreased body weight gains, and decreased food consumption.	U.S. EPA, 2006
			No data found.	
Rabbit	Oral	0, 62.5, 125, 250, 500 mg/kg/day	Maternal: increased abortion rates, decreased motor activity, and ataxia.	U.S. EPA, 2006
			Fetal death, increased abortion.	

B6-4.2.1.5 No Observable Adverse Effect Levels

Table B6-8 Mammalian NOAELs and LOAELs for Oral Eposure to Dicamba

Test Organism (Species)	Effect	Value (Mg/kg/day)	Endpoint	Reference
Sub-chronic				
Male Rat	NOAEL	479.4	Decreased body weight gains, increased liver weight and increased centrolobular hepatocyte hypertrophy and hepatocellular pigmentation	U.S. EPA, 2006
	LOAEL	1,000		
Female Rat	NOAEL	535.6		
	LOAEL	1065.3		
Chronic				
Male Mouse	NOAEL	358	Carcinogenesis	U.S. EPA, 2006
Female Mouse	NOAEL	354		
Male Rat	NOAEL	107	Carcinogenesis.	U.S. EPA, 2006
Female Rat	NOAEL	127		
Dog	NOAEL	52	No information found	U.S. EPA, 2006
Neurological				
Rat	LOAEL	300	Impaired respiration, rigidity upon handling, prodding, or dropping, impaired gait and righting reflex in both sexes.	U.S. EPA, 2006
Male Rat	NOAEL	401.4	Rigid body tone, slightly impaired righting reflex and gait.	U.S. EPA, 2006
	LOAEL	767		
Female Rat	NOAEL	472.0		
	LOAEL	1028.9		
Reproductive and developmental				
Pregnant Rat	NOAEL	160	Maternal: increased mortality rates, decreased body weight gains, decreased food consumption.	U.S. EPA, 2006
	LOAEL	400		
Rat pup	NOAEL	400	No information found	
	LOAEL	Not established		
Pregnant Rabbit	NOAEL	62.5	Maternal: increased abortion rates, decreased motor activity, ataxia.	U.S. EPA, 2006
	LOAEL	150		
Rabbit Pup	NOAEL	62.5	Fetal death, increased abortion rate.	
	LOAEL	150		
Parent systemic effects	NOAEL	122	Parental/Systemic: Slowing righting reflex. Reproductive: delayed sexual maturation in F1 females	U.S. EPA, 2006
	LOAEL	Male: 419		
		Female: 450		
Parent reproductive effects	NOAEL	122	Offspring: impaired pup growth, decreased pup weights	
	LOAEL	419	Parental/Systemic: Slowing righting reflex.	
Rat pup	NOAEL	45	Reproductive: delayed sexual maturation in F1 females	
	LOAEL	136		

B6-4.2.2 Dermal Exposure

B6-4.2.2.1 Death

Table B6-9 Mammalian Acute LD_{50} Value Resulting from Dermal Exposure to Dicamba

Test Organism (Species/Sex)	LD_{50} (mg/kg)	Reference
Acute		
Rat	≥2,000	U.S. EPA, 2006
Rat	>,2000	UNLM, 1995

B6-4.2.2.2 Systemic Effects

No data found.

B6-4.2.2.3 Neurological Effects

No data found.

B6-4.2.2.4 Reproductive/Developmental Effects

No data found.

B6-4.2.2.5 No Observed Adverse Effect Level

Table B6-10 Mammalian NOAELs and LOAELs for Dermal Exposure to Dicamba

Test Organism (Species)	Effect	Value	Endpoint	Reference
Sub-chronic				
Rat (m/f)	NOAEL	1,000 mg/kg/day	No information found	U.S. EPA, 2006

B6-4.2.3 Inhalation Exposure

B6-4.2.3.1 Death

Table B6-11 Mammalian LD_{50} Values resulting from Inhalation Exposure to Dicamba

Test Organism (Species/Sex)	LD_{50} (mg/L)	Reference
Acute		
Rat	≥5.3	U.S. EPA, 2006
Rat	>200	UNLM, 1995

B6-4.2.3.2 Systemic Effects

No data found.

B6-4.2.3.3 Neurological Effects

No data found.

B6-4.2.3.4 Reproductive/Developmental Effects

No data found.

B6-4.2.3.5 No Observed Adverse Effect Level

No data found.

Carcinogenicity

In 2006, the U.S. EPA classified Dicamba as "Not Likely to be Carcinogenic to Humans" by the oral route. Mutagenicity studies did not show evidence of any mutagenic potential for dicamba, although some positive results were reported in published literature (U.S. EPA, 2006).

Table B6-12 Mammalian Carcinogenicity Data

Test Subjects	Exposure	Dose	Response	Reference
Rat	Diet	Male: 0, 5.5, 17.2, 108, 358 mg/kg/day (unspecified period)	Not Carcinogenic. No other information found.	U.S. EPA, 2006
		Female: 0, 5.8, 18.8, 121, 354 mg/kg/day (unspecified period)		
Mouse	Diet	Male: 0, 5.5, 17.2, 108, 358 mg/kg/day (unspecified period)	Not Carcinogenic. No other information found.	U.S. EPA, 2006
		Female: 0, 5.8, 18.8, 121, 354 mg/kg/day (unspecified period)		

Structure Activity analysis of dicamba showed that its level of concern for carcinogenity is low (U.S. EPA, 2006). The U.S. EPA (2006) indicated that dicamba has been tested for mutagenicity and genotoxicity in microorganisms, drosophila, rats, and mammalian cell-cultures with both positive and negative results.

B6-4.3 Populations at Special Risk

No data found.

B6-4.4 Toxicokinetics

Dicamba is rapidly and extensively absorbed in rats following exposure through the oral route. It will be rapidly excreted predominantly as an unmetabolized compound in the urine (Durkin *et al*., 2004). Oral studies in rats have indicated that there were no significant pharmacokinetic differences in the dicamba free acid form and the dicamba amine salt forms (Durkin *et al*., 2004). The primary contaminant from technical grade dicamba manufacturing process is its 3,5-dichloro-isomer (Durkin *et al*., 2004). Dicamba did not accumulate in tissue. It is mainly excreted *via* the urine (96%) with a small percentage (4 %) excreted in the fecal matter (Durkin *et al*., 2004). Dicama is excreted mainly unchanged in the urine. However, approximately 20% will conjugate with glucuronic acid (Durkin *et al*., 2004).

After dermal application of dicamba, it was found that 14.1% of dicamba was absorbed through evaluating urinary excretion (Durkin *et al*., 2004). Highest blood concentrations of dicamba were found after the 1st and 9th hours respectively after dermal contact (Durkin *et al*., 2004). The disappearance of dicamba from blood will follow first order kinetics, as the half-life is only 0.4 hours (Durkin *et al*., 2004).

For risk assessment purposes the U.S. EPA (2006) used a dermal absorption factor of 15%.

B6-4.5 Exposure Limits

Table B6-13 Existing RfD Values for Dicamba Exposure

Reference Dose (mg/kg/day)	Route of Exposure	Reference	Endpoint	Study	Reference	NOAEL (mg/kg/day)	Uncertainty factor
Acute/Short-term (1-7 days)							
1.0	Oral	U.S. EPA, 2006	Impaired gaits and righting reflex	Acute Neurotoxicity Study in Rats	--	300[a]	300
Intermediate-term (7 days- Several months)							
No available values							
Long-term (6 months to lifetime)							
0.0125[b]	Oral	Health Canada, 1987, 2004	Hepatotoxicity, with vacuolization, necrosis, fatty deposits and liver weight changes.	Two-year feeding study in dogs	NAS, 1977	1.25	100
0.03	Oral	U.S. EPA, 1992	Maternal and fetal Toxicity	Rabbit Developmental Study	Velsicol Chemical Corp, 1978	3	100
0.45	Oral	U.S. EPA, 2006	Decreased pup weight	Multi-Generation Reproduction Study in Rats	--	45	100

[a] Used LOAEL to establish RfD, compensated by increasing the UF by 3x.

[b] 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.

Table B6-14 Summary of the Toxicological Dose and Endpoints for Dicamba used in Human Risk Assessment by the U.S. EPA (2006)

Exposure Scenario	Reference Dose	Endpoint	Study	NOEL (mg/kg/day)	LOEL (mg/kg/day)	LOC for MOE (residential and occupational)	Reference
Oral Short-term incidental (1-30 days)	0.45	Decreased pup weights	Multi-generation Reproduction Study in Rats	45	136	100	NR
Oral Intermediate-term incidental (1-6 months)	0.45	Decreased pup weights	Multi-generation Reproduction Study in Rats	45	136	100	NR
Dermal Short-term (1-30 days)	0.45	Decreased pup weights	Multi-generation Reproduction Study in Rats	45 (Dermal absorption rate = 15%)	136	100	NR
Dermal Intermediate-term (1-6 months)	0.45	Decreased pup weights	Multi-generation Reproduction Study in Rats	45 (Dermal absorption rate = 15%)	136	100	NR
Dermal Long-term (>6 months)	0.45	Decreased pup weights	Multi-generation Reproduction Study in Rats	45 (Dermal absorption rate = 15%)	136	100	NR
Inhalation Short-term (1-30 days)	0.45	Decreased pup weights	Multi-generation Reproduction Study in Rats	45 (inhalation absorption rate = 100%)	136	100	NR
Inhalation Intermediate-term (1-6 months)	0.45	Decreased pup weights	Multi-generation Reproduction Study in Rats	45 (inhalation absorption rate = 100%)	136	100	NR
Inhalation Long-term (>6 months)	0.45	Decreased pup weights	Multi-generation Reproduction Study in Rats	45 (inhalation absorption rate = 100%)	136	100	NR

NR Reference not provided

Based on the available general population (B6-13) and occupational reference doses (Table B6-14) the following exposure limits were selected for the risk assessment purposes of this report (Table B6-15).

Table B6-15 Summary of Selected TRVs for Dicamba

COC	TRV Type[a]	Route	TRV value (mg/kg/day)	Major Health Effects	Route of Exposure in Primary Study	Reference
Dicamba	Acute/Short-term RfD (1- 30 days)	Oral	0.45	Decreased pup weight	Oral	U.S. EPA, 2006
		Dermal				
		Inhalation				
	Intermediate-term RfD (1 to 6 months)	Oral	0.45	Decreased pup weight	Oral	U.S. EPA, 2006
		Dermal				
		Inhalation				
	Long-term RfD	Oral	0.45[a]	Decreased pup weight	Oral	U.S. EPA, 2006
		Dermal	0.45	Decreased pup weight	Oral	U.S. EPA, 2006
		Inhalation				

[a] Acceptable Daily Intake (ADI).

B6-5.0 ENVIRONMENTAL FATE AND EXPOSURE

B6-5.1 Air

Since dicamba is relatively volatile, it is readily dispersed in the environment. In a recent review, it was cited that the occurrence of dicamba, along with several other pesticides, in rainwater was found in sites distant from any known agricultural application (Majewski *et al*., 1995). In a small agricultural watershed in Canada, seasonal estimates of the atmospheric deposition of dicamba over a four-year period in the watershed ranged from 0.02 to 0.18% of the total amount applied each year (Waite *et al*. 1995).

The main route of dicamba degradation in the atmosphere is through reactions with electrophilic hydroxyl radicals (Mackay *et al*., 1997).

Table B6-14 Half-life of Dicamba in Air

Conditions	Half-life	Reference
Range half-lives	30-100 hours	Mackay *et al*., 1997
Mean half-life	55 hours	Mackay *et al*., 1997

B6-5.2 Water

The maximum acceptable concentration for dicamba in drinking water set by Health Canada is 0.12 mg/L (Health Canada, 1987).

Dicamba salts are highly soluble in water. Dicamba was found in 0.11 to 0.15% of the ground waters surveyed in the United States (USGS, 1998). The maximum level detected was 0.0025 mg/L. However, there was no apparent correlation between the prevalence of dicamba in groundwater from agricultural areas (0.11%) compared with non-agricultural urban areas (0.35%) (USDA/FS, 1999). Several additional studies reported higher frequencies of dicamba occurrence in groundwater from agricultural areas (Miller *et al*., 1995; Ritter *et al*., 1996).

Dicamba was detected in 0.32% of stream samples and 0.12% of samples from major aquifers in the United States (USGS, 1998). The highest level ever detected was 0.00016 mg/L(USGS 1998). In an agricultural area where herbicides are used extensively, dicamba was found in 17% to 55% of water samples from farm ponds and dugout waters (Grover *et al*., 1997). Dicamba was found in surface runoff when a rainstorm occurred soon after application to agricultural fields in western Washington (Mayer *et al.,* 1990). Several additional monitoring studies reported low concentrations of dicamba in soil runoff. However, usually percolation of dicamba through soil will predominate over soil runoff (USDA/FS, 1999). Dicamba was found in stream waters after aerial application to 166 acres (25%) of a Pacific Northwest forest watershed. Concentration rose to a maximum of 0.037 mg/L after 5.2 hours, and then dropped to background levels (<0.001 mg/L) after 37.5 hours.

Table B6-15 Half-life of Dicamba in Water

Conditions	Half-life	Reference
Range half-lives	300-1,000 hours	Mackay *et al*., 1997
Mean half-life	3 weeks	Mackay *et al*., 1997

B6-5.3 Sediment and Soil

Dicamba is highly mobile in and poorly adsorbed by most soil types (USDA/FS, 1999). The adsorption of dicamba to organo-clay soil is influenced by soil pH, with the greatest adsorption to soil occurring in acidic soils (Zhao *et al*., 1996). However, biodegradation of dicamba increases with temperature and soil moisture, with slightly faster degradation occurring in acidic soil (JW, 2005). Since dicamba is poorly adsorbed and highly soluble in water, it may contaminate groundwater supplies.

Dicamba is moderately persistent in soil. Its reported half-life in soil ranges from 1 to 6 weeks (USDA/FS, 1999). Dicamba is likely to be more rapidly degraded in soils with high microbial populations, but dissipates more slowly in hardwood forests and wetlands than would be expected from the results of laboratory studies (Voos *et al*., 1997a, b). The slower than expected field dissipation is probably attributable to sorption of dicamba in acidic and highly organic soil horizons (USDA/FS, 1999). In soil, dicamba breaks down to very simple substances like carbon dioxide and water (USDA/FS, 1999). Some intermediates structurally related to dicamba are formed during this process. One of the intermediates, 3,6-dichlorosalicylic acid, is adsorbed to soil much more strongly than dicamba (USDA/FS, 1999).

Table B6-16 Half-life of Dicamba in Soil

Conditions	Half-life	Reference
Range half-lives	300-1,000 hours	Mackay *et al*., 1997
Mean half-life	3 weeks	Mackay *et al*., 1997
Depend on soil conditions	1-4 weeks	JW, 2005
--	1-6 weeks	USDA/FS, 1999

B6-5.4 Other Environmental Media

Dicamba has a bioconcentration factor of 3.162 (JW, 2006).

B6-5.5 Plant Residues and Metabolism

Dicamba may cause damage to plants as a result of its absorption from the soil by plant roots (USDA/FS, 1999). The leaves and roots of plants rapidly absorb dicamba, and it is readily transported to other parts of plants (USDA/FS, 1999). It has been reported that in some plant species, dicamba will accumulate in the tips of mature leaves (Ahrens, 1994). Desirable broadleaf plants such as fruit trees and tomatoes may be harmed during their growth and development stages if dicamba is applied (Ahrens, 1994). Residues of dicamba will be excreted from the plants as exudates from the roots into the surrounding soil, or by loss from leaf surfaces (Ahrens, 1994).

B6-6.0 SUMMARY

Dicamba is an acid, and it will form salts in aqueous solutions. Different dicamba salts are formulated for herbicidal use and they include dimethylamine salt, sodium salt, isopropylamine salt, diglycolamine salt, and potassium salt. Being an auxin agonist, dicamba is commonly used for the control of annual and perennial broadleaf weeds, brush, and vines in rangeland and non-

cropland areas (USDA/FS, 1999). Between 1978 and 2000, approximately 30,000 kg of dicamba was applied over an area of 8,200 ha at CFB Gagetown (JW, 2006).

Most of the dicamba toxicity studies in rodents showed minimal effects even at the highest dosage levels tested. However, dicamba will produce neurological toxicity leading to adverse reproductive/developmental effects in rodents. The U.S. EPA (2006b) has classified dicamba as a non-human carcinogen.

B6-7.0 REFERENCES

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