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DICAMBA

DRAFT

Health Advisory
Office of Drinking Water
U.S. Environmental Protection Agency

I. INTRODUCTION

The Health Advisory (HA) Program, sponsored by the Office of Drinking Water (ODW), provides information on the health effects, analytical methodology and treatment technology that would be useful in dealing with the contamination of drinking water. Health Advisories describe nonregulatory concentrations of drinking water contaminants at which adverse health effects would not be anticipated to occur over specific exposure durations. Health Advisories contain a margin of safety to protect sensitive members of the population.

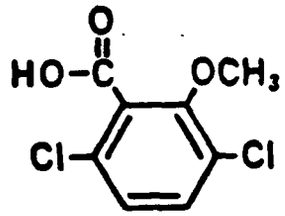
Health Advisories serve as informal technical guidance to assist Federal, State and local officials responsible for protecting public health when emergency spills or contamination situations occur. They are not to be construed as legally enforceable Federal standards. The HAs are subject to change as new information becomes available.

Health Advisories are developed for one-day, ten-day, longer-term (approximately 7 years, or 10% of an individual's lifetime) and lifetime exposures based on data describing noncarcinogenic end points of toxicity. Health Advisories do not quantitatively incorporate any potential carcinogenic risk from such exposure. For those substances that are known or probable human carcinogens, according to the Agency classification scheme (Group A or B), Lifetime HAs are not recommended. The chemical concentration values for Group A or B carcinogens are correlated with carcinogenic risk estimates by employing a cancer potency (unit risk) value together with assumptions for lifetime exposure and the consumption of drinking water. The cancer unit risk is usually derived from the linear multistage model with 95% upper confidence limits. This provides a low-dose estimate of cancer risk to humans that is considered unlikely to pose a carcinogenic risk in excess of the stated values. Excess cancer risk estimates may also be calculated using the One-hit, Weibull, Logit or Probit models. There is no current understanding of the biological mechani ms involved in cancer to suggest that any one of these models is able to predict risk more accurately than another. Because each model is based on differing assumptions, the estimates that are derived can differ by several orders of magnitude.

II. GENERAL INFORMATION AND PROPERTIES

CAS No. 1918-00-9

Structural Formula



3,6-Dichloro-2-methoxy-benzoic acid

Synonyms

- Banes, Banex, Banlen, Banuel D, Banvel, Brush buster, Dianat, Dianate, Dicambe, Mediben, Mondak, MDBA, Velsicol Compound R

Uses

- Herbicide used to control broadleaf weeds in field and silage corn, grain sorghum, small grains, asparagus, grass seed crops, turf, pasture, rangeland, and non-cropland areas such as fence rows, roadways and wastelands. For control of brush and vines in non-cropland, pasture and rangeland areas (Meister, 1983).

Properties (Berg, 1986; CHEMLAB, 1985; Meister, 1983; Windholz et al., 1983; Worthing, 1983)

Chemical Formula	$C_8H_6Cl_2O_3$
Molecular Weight	221.04
Physical State (at 25°C)	Crystals
Boiling Point	--
Melting Point	114 to 116°C
Density	--
Vapor Pressure (20°C)	3.75×10^{-3} mm Hg
Specific Gravity	--
Water Solubility (20°C)	6,500 mg/L at 25°C
Log Octanol/Water Partition Coefficient	3.67 (calculated)
Taste Threshold	--
Odor Threshold	--
Conversion Factor	--

Occurrence

- Dicamba has been found in 249 of 624 surface water samples analyzed and in 39 of 275 ground water samples (STORET, 1987). Samples were collected at 148 surface water locations and 229 ground water locations dicamba was found in 12 states. The 85th percentile of all non-zero samples was 0.15 ug/L in surface water and 0.07 ug/L in ground water.

The maximum concentration found in surface water was 3.3 ug/L, while in ground water it was 0.8 ug/L.

Environmental Fate

- In several aerobic soil metabolism studies, dicamba (acid or salt form not specified) had half-lives of 1 to 6 weeks in sandy loam, heavy clay, silty clay, clay loam, sand and silt loam soils at 18 to 38°C and 40 to 100% of field capacity. Degradation rates decreased with decreasing temperature and soil moisture (Smith, 1973a,b; Smith, 1974; Smith and Cullimore, 1975; Suzuki, 1978;1979).

- For the dimethylamine salt, half-lives in sandy loam and loam soils ranged from 17 to 32 days (Altom and Stritzke, 1973). Phytotoxic residues, detected by a non-specific bioassay method, have persisted in aerobic soil for almost 2 years (Sheets, 1964; Sheets et al., 1968).

- Based on soil thin-layer chromatography (TLC), dicamba (acid or salt form not specified) is highly mobile in sandy loam, silt loam, sandy clay loam, clay loam, loam, silty clay loam and silty clay soils (Helling, 1971; Helling and Turner, 1968).

- The free acid of dicamba and the dimethylamine salt were not appreciably adsorbed to any of five soils ranging from heavy clay to loamy sand (Grover and Smith, 1974). The dicamba degradation product, 3,6-dichlorosalicylic acid, adsorbed to sandy loam (30%), clay and silty clay (55%) (Smith, 1973a,b; Smith and Cullimore, 1975).

- Losses of 12 to 19% of the applied radioactivity from nonsterile soils indicated that metabolism contributes substantially more to ^{14}C-dicamba losses than does volatilization (Burnside and Levy, 1965; 1966).

- Under field conditions, dicamba (acid or salt form not specified) had half-lives of 1 to 2 weeks in a clay and a sandy loam soil when applied at 0.27 and 0.53 lb/A. At either application rate, less than 30 ppb of dicamba remained after 4 weeks (Scifres and Allen, 1973). In another study, using a nonspecific bioassay method of analysis, dicamba phytotoxic residues dissipated within 2 years in loam and silty clay loam (Burnside et al., 1971).

- Ditchbank field studies indicated vertical movement of dicamba in soil; the soil layers at 6 to 12 inches contained a maximum of 0.07 ppm and 0.28 ppm in canals treated at 0.66 and 1.25 lb/A, respectively (Salman et al., 1972).

III. PHARMACOKINETICS

Absorption

- Atallah and Yu (1980) reported that mice, rats, rabbits and dogs administered single oral doses of ^{14}C-dicamba (99% purity,

approximately). 100 mg/kg) excreted an average of 85% of the administered dose in urine in the 48 hours after dosing.

- Similar findings were reported for rats by Tye and Engel (1967) (96% excreted in 24 hours) and by Whitacre and Diaz (1976) (83% excreted in 24 hours). The data indicate that dicamba is rapidly absorbed from the gastrointestinal tract.

Distribution

- The retention of dicamba (99% purity, approximately 100 mg/kg) was investigated in rats, mice, rabbits and dogs following single doses by oral intubation (Atallah and Yu, 1980). Tissue levels 16 hours after treatment were low. Tye and Engel (1967) also found low residue levels of dicamba in kidneys, liver and blood. The data indicate that dicamba does not accumulate in mammalian tissues.

Metabolism

- The metabolism of ^{14}C-dicamba (99% purity) was investigated in mice, rats, rabbits and dogs after administration of single oral doses at approximately 100 mg/kg (Atallah and Yu, 1980). Between 97 to 99% of the dicamba was recovered unchanged in the urine of all four species. 3,6-Dichloro-2-hydroxybenzoic acid (DCHBA, a metabolite) was not detected in any urine sample at a level greater than 1% of the dose. There was also a small amount of unknown metabolites totaling about 1%.

Excretion

- Atallah and Yu (1980) investigated the excretion of ^{14}C-dicamba (99% purity) after a single oral dose (approximately 100 mg/kg) in mice, rats, dogs and rabbits, and reported that 67 to 93% of the administered dose was excreted in urine of the four species within 16 hours. The compound was found to a lesser degree in feces (0.5 to 5.7%) and various tissues (0.17 to 0.5%) 16 hours postdosing.

IV. HEALTH EFFECTS

Humans

- The Pesticide Incident Monitoring System data base revealed 10 incident reports involving humans from 1966 to March 1981 for dicamba alone (U.S. EPA, 1981). Six of the ten reported incidents involved spraying operations. No concentrations were specified. Exposed workers developed muscle cramps, dyspnea, nausea, vomiting, skin rashes, loss of voice or swelling of cervical glands. Four additional incidences resulted in coughing and dizziness in one child involved in an undescribed agricultural incident. Three children who sucked mint leaves from a ditch bank previously sprayed with dicamba were asymptomatic.

Animals

Short-term Exposure

- Reported acute oral LD_{50} values for technical dicamba [85.8% active ingredient (a.i.)] range from 757 to 1,414 mg/kg (Witherup et al., 1962) in rats. The acute oral LD_{50} in mice has been reported to be >4,640 mg/kg (Kettering Laboratory, 1962) and 316 mg/kg in hens (Roberts et al., 1983).

- An acute inhalation LC_{50} of >200 mg/L was reported in rats (IRDC, 1973).

- The neurotoxic effects of dicamba in hens were studied by Roberts et al. (1983). Technical dicamba (86.2% a.i.) was administered per os (10 hens/dose) in doses of 0, 79, 158 or 316 mg/kg. Two groups of ten hens each were dosed at 316 mg/kg. The various groups were observed for 21 days following treatment. No signs of ataxia were observed at any dose level tested. Histopathological evaluation of nervous tissue from 13 hens treated at 316 mg/kg demonstrated sciatic nerve damage in 6 hens (46%). The authors attributed this alteration to prolonged recumbency rather than a direct effect of dicamba. Based on the absence of delayed neurotoxicity and sciatic nerve damage, a NOAEL of 158 mg/kg is identified for this study.

- Rats (two/sex/dose) of the CD strain were fed diets containing 658 or 23,500 ppm of technical dicamba (85.8% a.i.) for up to three weeks (Witherup et al., 1962). Assuming that 1 ppm in the diet of rats is equivalent to 0.05 mg/kg/day (Lehman, 1959), these levels correspond to about 32.9 or 1,175 mg/kg/day. No adverse effects on physical appearance, behavior, food consumption, body or organ weights, gross pathology or histopathology were reported. Based on this information, a NOAEL of 1,175 mg/kg/day (the highest dose tested) is identified.

Dermal/Ocular Effects

- IRDC (1974) reported an acute LD_{50} of >2000 mg/kg in rabbit dermal studies.

- Heenehan et al. (1978) studied the sensitization potential of technical dicamba (86.8% a.i.) in albino guinea pigs. The compound was applied as a 10% suspension to the shaved backs of guinea pigs (five/sex) for 6 hours three times per week for 3 weeks. Following nine sensitizing doses, two challenge doses were applied. Dicamba was judged to cause moderate dermal sensitization.

- Technical dicamba (86.8% a.i.) was applied to the shaved backs of New Zealand White rabbits (four/sex/dose) in doses of 0, 100, 500 or 2,500 mg/kg/day, 5 days per week for 3 weeks (IRDC, 1979). Slight skin irritation was observed at 100 mg/kg, and moderate irritation at 500 mg/kg/day and above. No changes were observed in general appearance, behavior, body weight, organ weight, biochemistry, hematology or urinalysis.

- Thompson (1984) instilled single doses (0.1 g) of technical dicamba (purity not specified) into the conjunctival sacs of nine New Zealand rabbits; three eyes were washed and six were not washed. Dicamba was severely irritating and corrosive to both washed and unwashed eyes.

Long-term Exposure

- Laveglia et al. (1981) fed CD rats (20/sex/dose) technical dicamba (86.8% a.i.) in the diet for 13 weeks in doses of 0, 1,000, 5,000 or 10,000 ppm. Assuming that 1 ppm in the diet of rats is equivalent to 0.05 mg/kg/day (Lehman, 1959), this corresponds to doses of about 0, 50, 250 or 500 mg/kg/day. No compound-related effects were observed in general appearance, hematology, biochemistry or in urinalysis values, survival and gross pathology at any dose levels tested. There was an absence or reduction of cytoplasmic vacuolation of hepatocytes and a decrease in mean body weight for both sexes (6.3% in females and 7.5% in males) at 10,000 ppm (500 mg/kg/day). The body weight decrease was lower ($p < 0.05$) at week 13 when compared to controls. A NOAEL of 5,000 ppm (250 mg/kg/day) can be identified for this study.

- Male Wistar rats (20/dose) were fed diets containing technical dicamba at 0, 31.6, 100, 316, 1,000 or 3,162 ppm for 15 weeks (Edson and Sanderson, 1965). Assuming that 1 ppm in the diet of rats is equivalent to 0.05 mg/kg/day (Lehman, 1959), this corresponds to doses of about 0, 1.6, 5, 15.8, 50 or 158 mg/kg/day. Following treatment, general behavior, physical appearance, food consumption, organ weights, gross pathology and histopathology were evaluated. However, the authors presented data only for the evaluation of body and organ weights. Hematological, urinalysis or clinical chemistry studies were not reported. No adverse effects were observed in the parameters measured at 316 ppm (15.8 mg/kg/day) or less. Relative liver-to-body weight ratios increased (p value not specified) in at 1,000 and 3,162 ppm (50 and 158 mg/kg/day). Based on these data, the authors identified a NOAEL of 316 ppm (15.8 mg/kg/day).

- Davis et al. (1962) fed beagle dogs (three/sex/dose) technical dicamba (90% a.i.) in the diet in doses of 0, 5, 25 or 50 ppm for 2 years. Assuming that 1 ppm in the diet of dogs is equivalent to 0.025 mg/kg/day, (Lehman, 1959), this corresponds to doses of about 0, 0.125, 0.625 or 1.25 mg/kg/day. No compound-related effects were observed on survival, food consumption, hematology, urinalysis and organ weights. A decrease in body weight was observed in males at 25 and 50 ppm and in females at 50 ppm. No individual data except for body weight were reported, and no statistical evaluations were made. The authors did not present data on gross pathology. Histopathology was done only on the heart, lung, liver and kidney. Based on marginal information, a NOAEL of 5 ppm (0.125 mg/kg/day) can be identified.

- Sprague-Dawley rats (32/sex/dose) were fed technical dicamba (90% a.i.) in the diet for 2 years in doses of 0, 5, 50, 100, 250 or 500 ppm (Davis et al., 1962). Assuming that 1 ppm in the diet of rats is equivalent to 0.05 mg/kg/day (Lehman, 1959), this corresponds to doses of about 0, 0.25, 2.5, 5, 12.5 or 25 mg/kg/day. The authors

reported no adverse effects upon survival, body weight, food consumption, organ weight, hematologic values or histology at the dose levels tested. No data were presented for evaluation of pharmacologic effects, gross pathology, urinalysis or clinical chemistry. Incomplete histological data were presented. A NOAEL could not be determined for this study due to insufficient data.

Reproductive Effects

- Charles River CD rats (20 females or 10 males/dose) were fed diets containing technical dicamba (87.2% a.i.) in doses of 0, 5, 50, 100, 250 or 500 ppm through three generations (Kettering Laboratory, 1966). Assuming that 1 ppm in the diet of rats is equivalent to 0.05 mg/kg/day (Lehman, 1959), this corresponds to doses of about 0, 0.25, 2.5, 5, 12.5 or 25 mg/kg/day. Fertility index, gestation index, viability index, lactation index and pup development were comparable in treated and control rats. A NOAEL of 500 ppm (25 mg/kg/day) was identified.

Developmental Effects

- Technical dicamba (87.7% a.i.) was administered per os to pregnant New Zealand White rabbits (23-27/dose) at doses of 0, 1, 3 or 10 mg/kg/day from days 6 through 18 of gestation (IRDC, 1978). No maternal toxicity, fetotoxicity or teratogenic effects were observed at 1 and 3 mg/kg/day. There were slightly reduced fetal and maternal body weights and increased postimplantation losses in the 10 mg/kg/day dose group when compared to untreated controls. The author did not consider these differences to be statistically significant. The author identified a developmental toxicity NOAEL of 10 mg/kg/day (the highest dose tested). Based on a reduction in body weights and increased post-implantation losses at the highest dose, a maternal and fetotoxic NOAEL of 3 mg/kg/day was identified by EPA/OPP.

- Pregnant albino rats (20-24/dose) were administered technical-grade dicamba by gavage at dose levels of 0, 64, 160 or 400 mg/kg/day on days 6 throught 19 of gestation (Toxi Genetics, 1981). No maternal toxicity was observed up to 160 mg/kg/day. Dicamba-treated dams in the 400-mg/kg/day dosage group exhibited ataxia and reduced body weight gain; they consumed less food during the dosing period when compared with controls given vehicle alone ($p < 0.05$). No fetotoxicity or developmental effects were observed at the dose levels tested. Based on these findings, a NOAEL for maternal toxicity of 160 mg/kg/day is identified. The NOAEL for fetotoxic and developmental effects is 400 mg/kg/day (the highest dose tested).

Mutagenicity

- Moriya et al. (1983) reported that dicamba (up to 5,000 ug/plate) exhibited no mutagenic activity against Salmonella typhimurium (TA 98, TA 100, TA 1535, TA 1537 and TA 1538) or Escherichia coli (WP2 hcr) either with or without metabolic activation.

° An increased number of chromosomal aberrations ($p < 0.01$) were reported in mouse bone marrow cells exposed to 500 mg/kg dicamba (Kurinnyi et al., 1982). No other details were presented.

Carcinogenicity

° Sprague-Dawley rats (32/sex/dose) were administered dicamba (90% a.i.) in the diet for two years at doses of 0, 5, 50, 100, 250 or 500 ppm (Davis et al., 1962). Assuming that 1 ppm in the diet of rats is equivalent to 0.05 mg/kg/day (Lehman, 1959), this corresponds to doses of about 0, 0.25, 2.5, 5, 12.5 or 25 mg/kg/day. The treated rats did not differ from the untreated control animals with respect to the incidence, types and time of appearance of tumors.

V. QUANTIFICATION OF TOXICOLOGICAL EFFECTS

Health Advisories (HAs) are generally determined for one-day, ten-day, longer-term (approximately 7 years) and lifetime exposures if adequate data are available that identify a sensitive noncarcinogenic end point of toxicity. The HAs for noncarcinogenic toxicants are derived using the following formula:

$$HA = \frac{(NOAEL \text{ or } LOAEL) \times (BW)}{(UF) \times (\_\_\_ \text{ L/day})} = \_\_\_ \text{ mg/L } (\_\_\_ \text{ ug/L})$$

where:

NOAEL or LOAEL = No- or Lowest-Observed-Adverse-Effect-Level in mg/kg bw/day.

BW = assumed body weight of a child (10 kg) or an adult (70 kg).

UF = uncertainty factor (10, 100 or 1,000), in accordance with NAS/ODW guidelines.

___ L/day = assumed daily water consumption of a child (1 L/day) or an adult (2 L/day).

One-day Health Advisory

No information was found in the available literature that was suitable for determination of the One-day HA value for dicamba. Accordingly, it is recommended that the Ten-day HA value of 0.3 mg/L (calculated below) for a 10 kg child be used at this time as a conservative estimate of the One-day HA.

Ten-day Health Advisory

The developmental toxicity study by IRDC (1978) has been selected to serve as the basis for the Ten-day HA value for dicamba. In this study, pregnant rabbits administered technical dicamba (87.7 % a.i.) by gastric intubation at dosage levels of 1, 3 or 10 mg/kg/day from days 6 through 18

of gestation showed slightly reduced maternal body weights at 10 mg/kg/day. Similarly, fetal body weights were slightly reduced, and postimplantation losses were increased in the 10-mg/kg/day dose group.

Based on these data, a maternal and fetal toxicity NOAEL of 3 mg/kg/day is identified. A rat study (Toxi Genetics, 1981) of comparable duration determined higher maternal and fetal NOAELs (160 and 400 mg/kg/day, respectively).

The Ten-day HA for a 10-kg child is calculated as follows:

$$\text{Ten-day HA} = \frac{(3\ \text{mg/kg/day})\ (10\ \text{kg})}{(1\ \text{L/day})\ (100)} = 0.3\ \text{mg/L}\ (300\ \text{ug/L})$$

where:

3 mg/kg/day = NOAEL, based on absence of body weight loss and post-implantation losses.

10 kg = assumed body weight of a child.

100 = uncertainty factor, chosen in accordance with NAS/ODW guidelines for use with a NOAEL from an animal study.

1 L/day = assumed daily water consumption of a child.

Longer-term Health Advisory

No studies found in the available literature were suitable for determining a Longer-term HA value for dicamba. One 13-week rat study (Laveglia et al., 1981) and one 15-week rat study (Edson and Sanderson, 1965) reported NOAELs (250 mg/kg/day and 15.8 mg/kg/day, respectively) that were higher than the NOAEL (3 mg/kg/day) of the rabbit study (IRDC, 1978) selected to derive the Ten-day HA value. It is therefore recommended that the Reference Dose (RfD) derived below in the calculation of the Lifetime HA (0.0013 mg/kg/day) be used at this time as the basis for the Longer-term HA values. As a result, the Longer-term HA is 13 ug/L for the 10-kg child and is 50 ug/L for the 70-kg adult.

Lifetime Health Advisory

The Lifetime HA represents that portion of an individual's total exposure that is attributed to drinking water and is considered protective of noncarcinogenic adverse health effects over a lifetime exposure. The Lifetime HA is derived in a three step process. Step 1 determines the Reference Dose (RfD), formerly called the Acceptable Daily Intake (ADI). The RfD is an estimate of a daily exposure to the human population that is likely to be without appreciable risk of deleterious effects over a lifetime, and is derived from the NOAEL (or LOAEL), identified from a chronic (or subchronic) study, divided by an uncertainty factor(s). From the RfD, a Drinking Water Equivalent Level (DWEL) can be determined (Step 2). A DWEL is a medium-specific (i.e., drinking water) lifetime exposure level, assuming 100% exposure from that medium, at which adverse, noncarcinogenic health effects would not be expected to occur. The DWEL is derived from the multiplication of the RfD by the assumed body

weight of an adult and divided by the assumed daily water consumption of an adult. The Lifetime HA is determined in Step 3 by factoring in other sources of exposure, the relative source contribution (RSC). The RSC from drinking water is based on actual exposure data or, if data are not available, a value of 20% is assumed for synthetic organic chemicals and a value of 10% is assumed for inorganic chemicals. If the contaminant is classified as a Group A or B carcinogen, according to the Agency's classification scheme of carcinogenic potential (U.S. EPA, 1986), then caution should be exercised in assessing the risks associated with lifetime exposure to this chemical.

The 2-year dog study by Davis et al. (1962) has been selected to serve as the basis for deriving the Lifetime HA for dicamba. In this study, beagle dogs were administered technical dicamba at dietary levels of 0, 5, 25 or 50 ppm (0, 0.125, 0.625 or 1.25 mg/kg/day). A decrease in body weight was observed in males at 25 and 50 ppm and in females at 50 ppm. A NOAEL of 25 ppm (0.125 mg/kg/day) was identified.

The Lifetime HA is derived from this NOAEL as follows:

Step 1: Determination of the Reference Dose (RfD)

$$RfD = \frac{0.125 \text{ mg/kg/day}}{(100)} = 0.0013 \text{ mg/kg/day}$$

where:

0.125 mg/kg/day = NOAEL based on the absence of body weight loss.

100 = uncertainty factor, chosen in accordance with NAS/ODW guidelines for use with a NOAEL from an animal study.

Step 2: Determination of the Drinking Water Equivalent Level (DWEL)

$$DWEL = \frac{(0.0013 \text{ mg/kg/day})\ (70 \text{ kg})}{(2 \text{ L/day})} = 0.046 \text{ mg/L (46 ug/L)}$$

where:

0.0013 mg/kg/day = RfD.

70 kg = assumed body weight of an adult.

2 L/day = assumed daily water consumption of an adult.

Step 3: Determination of the Lifetime Health Advisory

$$\text{Lifetime HA} = (0.046 \text{ mg/L})\ (20\%) = 0.009 \text{ mg/L (9 ug/L)}$$

where:

0.046 mg/L = DWEL.

20% = assumed relative source contribution from water.

Evaluation of Carcinogenic Potential

- One study on the carcinogenicity of dicamba in rats has been reported; it revealed no evidence of carcinogenicity (Davis et al., 1962).
- The International Agency for Research on Cancer has not evaluated the carcinogenicity of dicamba.
- Applying the criteria described in EPA's guidelines for assessment of carcinogenic risk (U.S. EPA, 1986), dicamba is classified in Group D: not classified. This category is used for substances with inadequate evidence of carcinogenicity in animal studies.

VI. OTHER CRITERIA, GUIDANCE AND STANDARDS

- The NAS (1977) has calculated an ADI of 0.00125 mg/kg/day based on a NOAEL of 1.25 mg/kg/day from a 2-year feeding study in dogs and an uncertainty factor of 1,000. Assuming a body weight of 70 kg and a 20% source contribution factor, they calculated a Suggested-No-Adverse-Reaction-Level (SNARL) of 0.009 mg/L.
- Residue tolerances from 0.05 to 40 ppm have been established for a variety of agricultural products (U.S. EPA, 1985a).

VII. ANALYTICAL METHODS

- Analysis of dicamba is by a gas chromatographic (GC) method applicable to the determination of certain chlorinated acid pesticides in water samples (U.S. EPA, 1985b). In this method, approximately 1 L of sample is acidified. The compounds are extracted with ethyl ether using a separatory funnel. The derivatives are hydrolized with potassium hydroxide, and extraneous organic material is removed by a solvent wash. After acidification, the acids are extracted and converted to their methyl esters using diazomethane as the derivatizing agent. Excess reagent is removed, and the esters are determined by electron capture (EC) GC. The method detection limit for dicamba has been estimated to be 0.27 ug/L.

VIII. TREATMENT TECHNOLOGIES

- Available data indicate granular-activated carbon (GAC) adsorption to be a possible removal technique for dicamba.
- Whittaker et al. (1982) report that a reduction of pH from 7 to 3 increased the extent of dicamba GAC adsorption. No system performance was reported.

IX. REFERENCES

Atallah, Y.H., and C.C. Yu.* 1980. Comparative pharmacokinetics and metabolism of dicamba in mice, rats, rabbits and dogs. MRID 00128088.

Altom, J.D., and J.R. Stritzke. 1973. Degradation of dicamba, picloram, and four phenoxy herbicides in soils. Weed Sci. 21:556-560.

Berg, G.L. 1986. Farm Chemicals Handbook. Willoughby, OH: Meister Publishing Co.

Burnside, O.C., and T.L. Levy. 1965. Dissipation of dicamba. Unpublished study prepared by the University of Nebraska, Department of Agronomy, submitted by Velsicol Chemical Corporation, Chicago, Ill.

Burnside, O.C., and T.L. Levy. 1966. Dissipation of dicamba. Weeds 14:211-214.

Burnside, O.C., G.A. Wicks and C.R. Fenster. 1971. Dissipation of dicamba, picloram, and 2,3,6-TBA across Nebraska. Weed Sci. 19:323-325.

CHEMLAB. 1985. The Chemical Information System, CIS, Inc.

Davis, R.K., W.P. Jolly, K.L. Stemmer et al.* 1962. The feeding for two years of the herbicide 2-methoxy-3,6-dichlorobenzoic acid to rats and dogs. MRID 00028248.

Edson, E.F., and D.M. Sanderson. 1965. Toxicity of the herbicides 2-methoxy-3,6-dichlorobenzoic acid (Dicamba) and 2-methoxy-3,5,6-trichlorobenzoic acid (tricamba). Food Cosmet. Toxicol. 3:299-304.

Grover, R., and A.E. Smith. 1974. Adsorption studies with the acid and dimethylamine forms of 2,4-D and dicamba. Can. J. Soil Sci. 54:179-186.

Heenehan, P.R., W.E. Rinehart and W.G. Brun.* 1978. A dermal sensitization study in guinea pigs. Compounds: Banvel 45, Banvel technical: Project No. 5055-78. MRID 00023691.

Helling, C.S. 1971. Pesticide mobility in soils: II. Applications of soil thin-layer chromatography. Soil Sci. Soc. Amer. Proc. 35:737-748.

Helling, C.S., and B.C. Turner. 1968. Pesticide mobility: Determination of soil thin-layer chromatography. Science. 162:562-563.

IRDC.* 1973. International Research and Development Corporation. Acute inhalation exposure in the male albino rats. Report No. 163-191. MRID 00028234.

IRDC.* 1974. International Research and Development Corporation. I. Acute toxicity studies in rats and rabbits. Report No. 163-295. MRID 00025372.

IRDC.* 1978. International Research and Development Corporation. Teratology study in rabbits. Report No. 163-436. MRID 00025373.

IRDC.* 1979. International Research and Development Corporation. Three-week dermal toxicity study in rabbits. Report No. 163-620. MRID 00128090.

Kettering Laboratory.* 1966. The effects exerted upon the fertility of rats and upon the viability of their offspring by the introduction of Banvel D into their diets. MRID 00028249.

Kettering Laboratory.* 1962. The cumulative toxicity of 2-methoxy-3,6-dichlorobenzoic acid (Banvel D) and 2-methoxy-3,5,6-trichlorobenzoic acid (Banvel T) when fed to rats. MRID 00022503.

Kurinnyi, A.I., M.A. Pilinskaya, I.V. German and T.S. L'vova. 1982. Implementation of a program of cytogenetic study of pesticides: Preliminary evaluation of cytogenetic activity and potential mutagenic hazard of 24 pesticides. Tsitol. Genet. 16:45-49.

Laveglia, J., D. Rajasekaran, L. Brewar.* 1981. Thirteen week dieting toxicity study in rats with dicamba. IRDC No. 163-671. MRID 00128093.

Lehman, A.J. 1959. Appraisal of the safety of chemicals in foods, drugs and cosmetics. Assoc. Food Drug Off. U.S.

Meister, R., ed. 1983. Farm Chemicals Handbook. Willoughby, OH: Meister Publishing Co.

Moriya, M., T. Ohta, K. Watanabe, T. Miyazawa, K. Kato and Y. Shirasu. 1983. Further mutagenicity studies on pesticides in bacterial reversion assay systems. Mutat. Res. 116:185-216.

NAS. 1977. National Academy of Sciences. Drinking Water and Health. Washington, DC: National Academy of Science Press.

Roberts, N., C. Fairley, C. Fish et al.* 1983. The acute oral toxicity (LD_{50}) and neurotoxicity effects of dicamba in the domestic hen. HRC Report No. 24/8355. MRID 00131290.

Salman, H.A., T.R. Bartley and A.R. Hattrup. 1972. Progress report of residue studies on dicamba for ditchbank weed control. U.S. Department of the Interior, Bureau of Reclamation, Applied Sciences Branch, Division of General Research, Engineering and Research Center. USDI, Br. Report No. REC-ERC-72-6; available from National Technical Information Center, Springfield, VA. 22151.

Scifres, C.F., and T.J. Allen. 1973. Dissipation of dicamba from grassland soils of Texas. Weed Sci. 21:393-396.

Sheets T.J. 1964. Letter sent to Warren H. Zick dated Jan.3, 1964. Greenhouse persistence study with dicamba and tricamba. U.S. Agricultural Research Service, Crops Research Division, Crops Protection Research Branch, Pesticide Investigations--Behavior in soils; unpublished study.

Sheets, T.J., J.W. Smith and D.D. Kaufman. 1968. Persistence of benzoic and phenylacetic acids in soils. Weed Sci. 16:217-222.

Smith, A.E. 1973a. Degradation of dicamba in prairie soils. Weed Res. 13:373-378.

Smith, A.E. 1973b. Transformation of dicamba in Regina heavy clay. J. Agric. Food Chem. 21:708-710.

Smith, A.E. 1974. Breakdown of the herbicide dicamba and its degradation products 3,6-dichlorosalicylic acid in prairie soils. J. Agric. Food Chem. 22:601-605.

Smith, A.E., and D.R. Cullimore. 1975. Microbiological degradation of the herbicide dicamba in moist soils at different temperatures. Weed Res. 15:59-62.

STORET. 1987.

Suzuki, H.K. 1978. Dissipation of Banvel and in combination with other herbicides in two soil types: Report NO. 196. Unpublished study prepared in cooperation with International Research and Development Corporation, submitted by Velsicol Chemical Corporation, Chicago, Ill.

Suzuki, H.K. 1979. Dissipation of Banvel or Banvel in combination with other herbicides: Two soil types: Report No. 197. Unpublished study prepared in cooperation with Craven Laboratories, Inc.; submitted by Velsicol Chemical Corporation, Chicago, Ill.

Thompson, G.* 1984. Primary eye irritation study in albino rabbits with technical dicamba. Study No. Will 15134. Will Research Laboratories, Inc. MRID 00144232.

Toxi Genetics.* 1981. Teratology study in albino rats with technical dicamba. Study No. 450-0460. MRID 00084024.

Tye, R., and D. Engel. 1967. Distribution and excretion of dicamba by rats as determined by radiotracer technique. J. Agric. Food Chem. 15:837-840.

U.S. EPA. 1981. U.S. Environmental Protection Agency. Summary of reported incidents involving dicamba. Pesticide incident monitoring system. Report No. 432. Office of Pesticide Programs, Washington, DC.

U.S. EPA. 1985a. U.S. Environmental Protection Agency. Code of Federal Regulations. 40 CFR 180.227. July 1, 1985.

U.S. EPA. 1985b. U.S. Environmental Protection Agency. U.S. EPA Method 615 - Chlorinated phenoxy acids. Fed. Reg. 50:40701, October 4, 1985.

U.S. EPA. 1986. U.S. Environmental Protection Agency. Guidelines for carcinogen risk assessment Fed. Reg. 51(185):33992-34003. September 24.

Whitacre, D.M. and L.I. Diaz.* 1976. Metabolism of ^{14}C-dicamba in female rats. MRID 00025363.

Whittaker, K.F., J.C. Nye, R.F. Weekash, R.J. Squires, A.C. York and H.A. Razemier. 1982. Collection and treatment of wastewater generated by pesticide application. U.S. Environmental Protection Agency. EPA-600/2-82-028, Office of Environmental Criteria and Assessment, Cincinnati, Ohio.

Windholz, M., S. Budavari, R.F. Blumetti, E.S. Otterbein, eds. 1983. The Merck Index -- An Encyclopedia of Chemicals and Drugs, 10th ed. Rahway, NJ: Merck and Company, Inc.

Witherup, S., K.L. Stemmer and H. Schlect.* 1962. The cumulative toxicity of 2-methoxy-3,6-dichlorobenzoil acid (Banvel D) and 2-methoxy-3,5,6-trichlorobenzoil acid (Banvel T) when fed to rats. MRID 00022503.

Worthing, C.R, ed. 1983. The Pesticide Manual: A World Compendium, 7th Ed. London: BCPC Publishers.

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