SEGi Review ISSN 1985-5672
Vol. 5, No. 1, July 2012, 5-13
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A RISK ASSESSMENT OF THE HERBICIDE *DICAMBA*

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ABSTRACT

Dicamba selectively kills some plant species and not others. Resistant plants survive by detoxifying the herbicide or not absorbing it. A resistant crop plant will be more tolerant to herbicide than the weeds around it. A major problem with Dicamba is its extreme leaching property in soils, regardless of organic matter or clay content. It also has high water solubility. Dicamba residues in food do not pose a hazard to the consumer provided the risk exposure does not exceed the limit. In the case of the European Union, the Theoretical Maximum Daily Intake, for a 70 kg adult is < 1.1 % of the Acceptable Daily Intake. Additional intakes of water and products of animal origin are not expected to give rise to problems. Estimates of acute dietary exposure of adults and toddlers revealed that the Acute Reference Dose would not be exceeded. The level of human exposure should be less than 0.1 mg/kg, 3000 times less than the Low Observed Adverse Effect Level (LOAEL) of 300 mg/kg (USDA 1999). The potential cancer risk calculated by the US EPA is in the 10^{-8} range, well below the action level of 10^{-6} for nitrosamines and other carcinogens.

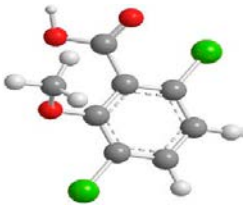
1.0 Introduction

Manufactured by Sandoz Crop Protection of Switzerland, *Dicamba* is a benzoic acid herbicide registered with the EPA in 1967 by Velsicol Chemical Corp. *Dicamba* attracted attention because of the toxicity of its contaminants, both dioxin and nitrosamines, and its propensity to leach through soil (Beyond Pesticides 2007). The herbicide mimics the plant hormone auxin, causing uncontrolled growth which eventually kills plants (NPIC). It is meant as post-emergent herbicide for controlling a broad spectrum of woody plants and certain annual, biennial and perennial broadleaf weeds in grain crops, grasslands, brush and bracken in pastures in combination with a phenoxyalkanoic acid or other herbicide (Hayes 2010a). It is also used on turf, rights-of-way, pastures, seed-crops, soybeans, wheat, oats, barley, asparagus, and sugarcane to control woody plants and broadleaf weeds (Beyond Pesticides 2007; Pound and Street 2010) before or after weed emergence (Hayes 2010a). *Dicamba* is systemic in nature and legumes can be killed (Extoxnet 1996) through absorption by the plant leaves and by the roots in the soil (Hayes 2010a). It has a variety of acid and salt formulations and is commercially available in liquids, liquid concentrates, wettable powders, granules, water dispersible granules, and sprays (Thoreby 2010). Its salts include isopropylamine, diglycoamine, dimethylamine, potassium, and sodium (NPIC 2010). *Dicamba* (3,6-dichloro-2-methoxybenzoic acid) is frequently formulated with other active herbicides such as 2,4-D, MCPP, and MCPA (NPIC). It may be sold under names such as Banfel, Banvel, Banvel CST, Banvel D, Banvel XG, Dianat, Dicazin, Fallowmaster, Mediben, Metambane, Tracker, and Trooper (Extoxnet 1993; Thoreby 2010) and Vanguish (Hayes 2010a; Wikipedia 2010). USDA (1999) has a list of *Dicamba* acid and/or salts such as Banvel (as the DMA salt 48.2%, DMA salts of related acids 12.0%, inert ingredients 39.8%) and Vanquish (as the DGA salt 56.8%, DGA salts of

related acids 14.2%, inert ingredients 29.0%) for forestry and non-crop sites supplied by BASF (USDA 1999).

Dicamba selectively kills some plant species but not others because resistant plants (James *et al*. 2005) survive by detoxifying the herbicide or not absorbing it so a crop plant will be more tolerant to the herbicide than the weeds around it. It does not injure grasses at recommended application rates (USDA 1999), so users apply it during pre- or post-emergence to control broadleaf weeds (NPIC 2010).

Molecular Structure of *Dicamba* (NPIC)



In January 2009, BASF SE and Monsanto Company announced a new joint-licensing agreement to accelerate the development of the next-generation of *Dicamba*-based weed control chemistry products. Both parties would participate in the development of innovative formulations for use with herbicide-resistant cropping systems. Improved formulations of *Dicamba* were being developed to complement a combination of herbicide-resistant crops. BASF is currently the largest provider of *Dicamba* and *Dicamba*-based products (Kissling 2009).

2.0 Methods: Residue Assay

The most common method used to detect and analyze *Dicamba* in water, soil, and biological material involves column/gas-liquid chromatography. The lowest reported limit of detection for *Dicamba* in water was 0.03 ppb with recovery rates ranging from 81.2% to 95% (USDA 1999). Pure *Dicamba* is an odourless, white crystalline solid and soluble in water (NPIC), and it boils at 200°C (Thoreby 2010) while the technical acid is a pale buff crystalline solid resistant to hydrolysis and oxidation under normal conditions (Extoxnet 1993).It is stable under normal temperatures and pressures, but it may pose a slight fire hazard if exposed to heat or flame in the presence of strong oxidizers as thermal decomposition releases toxic and corrosive fumes of chlorides and toxic oxides of carbon that could be a drift hazard (Extoxnet 1993; Kegley *et al*. 2010).

3.0 Risk Assessment of a Hazardous Agent

A risk is the probability that an adverse outcome will occur in a person or group that is exposed to a particular dose of a hazardous agent. The process used to estimate the likelihood that a person's health will be affected adversely under a specific set of conditions is called risk assessment (Hayes 2010b).

4.0 Hazard Identification

Symptoms and signs of poisoning with *Dicamba* include loss of appetite (anorexia), vomiting, muscle weakness, slowed heart rate, shortness of breath, central nervous system effects (victim may become excited or depressed), benzoic acid in the urine, incontinence,

cyanosis (bluing of the skin and gums),and exhaustion following repeated muscle spasms (Hayes 2008; WD 2010). In addition to these symptoms, inhalation can cause irritation of the linings of the nasal passages and the lungs, and loss of voice. Most individuals who have survived poisoning from *Dicamba* have recovered within 2 to 3 days with no permanent effects. (Hayes 2010a). It is a very irritating and corrosive agent, and can cause severe and permanent damage to the eyes. The eyelids may swell and the cornea may be cloudy for a week after *Dicamba* is splashed into the eyes. The eyes should be flushed with running water for at least 15 minutes if it is splashed into them. In some individuals, it is a skin sensitizer and may cause skin burns (NPIC 2010). There is no evidence that it is absorbed into the body through the skin (Extoxnet 1996). Dermal and inhalation exposure to humans may occur during application, particularly via splashing during dilution, mixing, and loading. Spraying of the herbicide by aircraft increases the potential for exposure to humans, livestock, and wildlife due to spray drift and ventilation (Hayes 2010b).

5.0 Tests to Determine Cause of Adverse Health Effects

5.1 LD50/LC50

A common measure of acute toxicity is the lethal dose (LD50) or lethal concentration (LC50) that causes death (resulting from a single or limited exposure) in 50 percent of the treated animals. LD50 is generally expressed as the dose in milligrams (mg) of chemical per kilogram (kg) of body weight. LC50 is often expressed as mg of chemical per volume (e.g., liter (L)) of medium (i.e., air or water) the organism is exposed to. Chemicals are considered highly toxic when the LD50/LC50 is small and practically non-toxic when the value is large. However, the LD50/LC50 does not reflect any effects from long-term exposure (i.e., cancer, birth defects, or reproductive toxicity) that may occur at levels below those that cause death (NPIC 2010). A reference on toxicity category is listed by NPIC (2010) below:

Figure 1: Toxicity Category (Adopted from NPIC 2010)

	High Toxicity (*Danger*)	Moderate Toxicity (*Warning*)	Low Toxicity (*Caution*)	Very Low Toxicity (*Caution*)
Oral LD50	Less than 50 mg/kg	50 - 500 mg/kg	500 - 5000 mg/kg	Greater than 5000 mg/kg
Dermal LD50	Less than 200 mg/kg	200 - 2000 mg/kg	2000 - 5000 mg/kg	Greater than 5000 mg/kg
Inhalation LC50	Less than 0.05 mg/l	0.05 - 0.5 mg/l	0.5 - 2 mg/l	Greater than 2 mg/l
Eye Effects	Corrosive	Irritation persisting for 7 days	Irritation reversible within 7 days	Minimal effects, gone within 24 hrs
Skin Effects	Corrosive	Severe irritation at 72 hours	Moderate irritation at 72 hours	Mild or slight irritation

5.2 Test on Species

Results showed that *Dicamba* was capable of inducing DNA damage since it significantly increased the unwinding rate of rat liver DNA in vivo (at 26.6 and 17.8 mg/kg body weight) and also induced UDS in HPBL in vitro in the presence of exogenous metabolic activation (S-9 mix) (Hayes 2010a). In a 3-generation study, *Dicamba* did not affect the reproductive capacity of rats. When rabbits were given doses of 0, 0.5, 1, 3, 10 or 20 (mg/kg)/day of technical *Dicamba* from days 6 through 18 of pregnancy, toxic effects on the mothers, slightly reduced fetal body weights, and increased loss of fetuses occurred at

the 10 mg/kg dose. In dog tests, some enlargement of liver cells had occurred, but a similar effect had not been shown in man (Wikipedia 2010; Wood 2010). U.S. EPA has set the no-observed adverse effect level (NOAEL) for this study at 3 (mg/kg)/day. NOAEL refers to the chemical dose at which no statistically or biologically significant increases in frequency or severity of adverse effects are observed between the exposed population and its appropriate control. Effects may be produced at this dose, but they are not considered to be adverse (USDA 1999). *Dicamba* is suspected of being a human teratogen. No teratogenic effects have been shown in lab animals such as rabbits and rats; it also has not been shown to be a mutagen (Wikipedia 2010; Cummins 2007). According to a 1983 Registration Standard, *Dicamba* is of low acute toxicity by either the oral or dermal routes, but is quite irritating to the eyes. It is rapidly excreted in urine, mostly as the unchanged compound and as 3,6-dichloro-2-hydroxybenzoic acid, also known as 3,6-dichloro-salicylic acid. Studies indicate that residues do not bioaccumulate in biological systems. Based on available industry data, EPA does not consider it to be either a reproductive toxin or to cause birth defects and preliminary short term testing has not indicated any mutagenic activity. It is absorbed by the leaves and translocated throughout the plant, where it exerts an auxin-like growth regulatory effect. Soybeans are extremely sensitive to it and toxic effects have been shown to occur not only from drift, but also from volatilization from the surfaces of treated leaves in neighbouring fields (Beyond Pesticides 2007).

5.3 Effects to Human Population

A major problem with *Dicamba* is its extreme mobility (leaching) in soils, regardless of organic matter or clay content, and high water solubility. USDA found that *Dicamba* was the most mobile of forty herbicides evaluated, and warned that it would likely contaminate groundwater (Beyond Pesticides 2007). Its half-life of in soil has been observed to vary from 4 to 555 days with a typical half-life of 1 to 4 weeks. Under conditions suitable to rapid metabolism, the half-life is less than two weeks. If released to water, microbial degradation appears to be the important *Dicamba* removal process; photolysis may contribute to its removal from water. Aquatic hydrolysis, volatilization, adsorption to sediment, and bioconcentration are not expected to be significant. If released to the atmosphere, it will probably exist in the vapour phase and be adsorbed into the particulate phase. The half-life for the vapour-phase reaction with photochemically produced hydroxyl radicals has been estimated to be 6 days. In the particulate phase it is subject to wet and dry deposition (Hayes 2010a). It bonds with soil poorly.Its half-life of in soil ranges from 1 to 6 weeks (USDA 1999; Extoxnet 1996).

An example on half life degradation is as follows:
1 half-life = 50% degraded; 2 half-lives = 75% degraded; 3 half-lives = 88% degraded;
4 half-lives = 94% degraded; 5 half-lives = 97% degraded

6.0 The Data

6.1 Chemical Structures

Dicamba (CAS registry number: 1918-00-9) has the empirical formula $C_8H_8Cl_2O_3$ and a molecular weight of 221.04. The pure material is a white crystalline solid with a melting point of 87-108 C, and is slightly soluble in water (50g/100 ml) but soluble in most organic solvents and melts at temperatures between 90 and 100 °C (194 to 212 °F). It is nonflammable and does not present any unusual handling hazards. (Hayes 2010a).

6.2 *In Vivo* Data - Animal Studies

The manufacturer, Sandoz Crop Protection, sponsored a combined chronic toxicity/oncogenicity two-year feeding study in rats that revealed a NOAEL of at least 500 ppm, the highest dose administered. A similar study was conducted in dogs that revealed a NOAEL of 50 ppm. These studies were conducted by Industrial BioTest. Witherup and Cleveland (1962) reported that rats fed on diets containing 5, 50, 100, 250 or 500 ppm and dogs fed on diets containing 5, 25, or 50 ppm of *Dicamba* for two years survived and showed no signs of intoxication or other adverse effects (Hayes 2010a). It is low in toxicity when ingested. The acute oral LD50 in rats is >2740 mg/kg based on the laboratory animals fed with high doses to cause toxic effects to help scientists judge overexposure. When pesticide products are used according to the label directions, toxic effects are not likely to occur because the amount of pesticide that people and pets may be exposed to is low compared to the doses fed to laboratory animals (NPIC 2010). In a 13-week oral study, investigators exposed male and female mice to *Dicamba* at approximate doses of 0, 500, 1000, 1250, or 1500 mg/kg/day. At doses of 1000 mg/kg/day and higher, they noted altered liver cells and lower body weights and reduced food consumption. The NOAEL was 500 mg/kg/day (NPIC 2010).

6.3 Human Studies

In oral, dermal, and inhaled routes of exposure, *Dicamba* has a low acute toxicity (NPIC). It may have irritating or corrosive effects on the skin and eyes (NPIC). PANNA reported that the EPA had identified it as a developmental toxin in the Toxics Release Inventory.

6.4 Occupational Exposure

No occupational exposure limits have been established for *Dicamba* by OSHA, NIOSH or ACGIH (Extoxnet 1993). The manufacturing process for *Dicamba* has the potential of resulting in traces [50 parts per billion (ppb)] of 2, 7-dichlorodibenzo-p-dioxin as a contaminant. The more toxic dioxin isomer 2, 3, 7, 8-tetrachlorodibenzo-p-dioxin (TCDD) has not been found at the limit of detection (2 ppb) and is not expected to be an impurity in *Dicamba*. The technical formulation also contains low levels of a carcinogenic impurity, dimethyl- N-nitrosoamine. Nitrosamine levels in the formulations are expected to be less than 1 ppm. (Hayes 2010a). With a vapour pressure of 3.4×10^{-5} mm Hg at 25 deg C, if released to the atmosphere will probably exist in both the vapour-phase and the adsorbed in the particulate phase. The half-life for the vapour-phase reaction with photochemically produced hydroxyl radicals has been estimated to be 2.42 days. The particulate phase will be subject to wet and dry deposition. It has a half-life of 31 days with a first-order rate constant of 0.0224/day in a typical mid-western agricultural soil under aerobic conditions. Its half-life in the same soil after the soil is made anaerobic at 30 days is 58 days with a first-order rate constant of 0.012/day. It is completely mineralized to CO2 under aerobic conditions with 3, 6-dichlorosalicylic acid as the only major metabolite. Low levels of 2, 3-dihydroxy-3, 6-dichlorosalicylic acid have been detected. Metabolism under anaerobic conditions is similar to that which occurred in aerobic soil except the rate of its metabolism is reduced under anaerobic conditions. (Hayes 2010a).

6.5 Risk Characterization

The toxicity assessments could estimate the likelihood of an adverse health impact under various conditions of exposure. It can be described in RITE (Risk =Toxicity x Exposure) as follows:

Toxicity (Hazard) $$ADI = \frac{NOAEL}{\text{Safety factors (Uncertainty Factors)}}$$

Definition:
ADI = Acceptable Daily Intake ingested daily throughout life without appreciable health risk.

NOAEL = No observed adverse effect level derived from safety studies usually in animals, which identify the hazard and the most sensitive species.

Uncertainty Factors = Factor applied to allow for differences between animals and humans, and between different humans. Assumptions made under unavailability of data.

In the EC (2008) Standing Committee, a review had been proposed by the Member States using following reference values for *Dicamba*:

ADI: 0.3 mg/kg bw/day; ARfD: 0.3 mg/kg bw/day; AOEL: 0.3 mg/kg bw/day
* bw = body weight

With particular regard to residues, the review has established that application consistent with good plant protection practice have no harmful effects on human or animal health. The Theoretical Maximum Daily Intake (TMDI; excluding water and products of animal origin) for a 70 kg adult is < 1.1 % of the Acceptable Daily Intake (ADI), based on the UK diet (UK DOE 2005). Additional intakes from water and products of animal origin are not expected to give rise to problems. Estimates of acute dietary exposure of adults and toddlers revealed that the Acute Reference Dose (ARfD) would not be exceeded (European diet - < 2 % for adults, schoolchildren or infants).

$$TDI + \frac{Ci \times CRi}{BW}$$

$$\text{Exposure} + \frac{C \times IR \times B}{BW \times AT}$$

*Ci = concentration in exposure media. Ci in food (mg.kg); Cri = rate of ingestion of food (g/day); B= bioavailability; IR=ingestion rate; AT= at time over does average; C=exposure concentration

The USDA acute oral RfD: For assessing the consequences of acute dietary exposure to *Dicamba*, the U.S. EPA uses a LOAEL for neurotoxicity of 300 mg/kg and recommends a margin of exposure (MOE) of 3000. In other words, the level of human exposure should be less than 0.1 mg/kg, 3000 times less than the LOAEL of 300 mg/kg (USDA 1999).

$$\text{Acute oral RfD MOE} = \frac{\text{LOAEL (mg/kg/day)}}{\text{Human Exposure (mg/kg/day)}}$$

MOE = 3000 = 300/ 0.1

Hayes (2010a) reported that combined chronic toxicity/oncogenicity two-year feeding study in rats revealed a NOAEL of 500 ppm, the highest dose administered. The study has been repeated with the same negative results. The data have been evaluated by the US EPA.

The potential oncogenic risk was calculated by the EPA to be in the 10^{-8} range, well below the US EPA action level of 10^{-6} for nitrosamines and other carcinogens (Hayes 2010a).

Cancer Risk (CR) = P (d) = Exposure x Cancer Slope Factor
Where, the target P (d) = 1 x 10^{-8}

Result is below EPA 1 x 10^{-6} shows lack of carcinogenicity.

$$\text{Alternatively cancer risk MOE} = \frac{\text{NOAEL (mg/kg/day)}}{\text{Human Exposure (mg/kg/day)}}$$

Quantifying Non-Carcinogenic hazards by using Hazard Quotient (HQ):

$$\text{HQ} = \frac{\text{Intake}}{\text{RfD or RfC or ADI}}$$

* A Hazard Quotient of <1 is typically considered to be of little concern for policy decision.

7.0 Conclusion

Dicamba residues in food do not pose a hazard to the consumer if the risk exposure does not exceed the limit. In the case of EC (2008), the TMDI excluding water and products of animal origin) for a 70 kg adult is < 1.1 % of the Acceptable Daily Intake (ADI), based on the UK diet (UK DOE 2005). Additional intake from water and products of animal origin are not expected to give rise to problems. Estimates of acute dietary exposure of adults and toddlers revealed that the Acute Reference Dose (ARfD) would not be exceeded (European diet - < 2 % for adults, schoolchildren or infants). For assessing the consequences of acute dietary exposure to *Dicamba*, the U.S. EPA uses a LOAEL for neurotoxicity of 300 mg/kg and recommends a margin of exposure (MOE) of 3000. In other words, the level of human exposure should be less than 0.1 mg/kg, 3000 times less than the LOAEL of 300 mg/kg (USDA 1999). The potential cancer risk calculated by the EPA to be in the 10^{-8} range, well below the US EPA action level of 10^{-6} for nitrosamines and other carcinogens shows lack of carcinogenicity effect. The Final Environmental Impact Statement (FEIS) for Managing Competing and Unwanted Vegetation (Durkin and Bosch, 1988) predicts levels of human exposure (dose) for project workers and for the public, for both a typical field project and for a large accidental spill. These dose levels are compared with the highest dose level in animal tests that showed No Observed Effects Level (NOEL). This level of exposure is referred to as the Margin of Safety (MOS) or Margin of Exposure approach (USDA 1999). NOEL refers to the chemical dose at no treatment-related effects were observed while

MOS refers to the ratio between an effect or no effect level in an animal and the estimated human dose.

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