Environmental Protection Agency
Office of Pesticide Programs (TS-766C)
Washington, DC 20460

EPA

Pesticide Fact Sheet

Name of Chemical: Dichlobenil
Reason for Issuance: Registration Standard
Date Issued: MAR 23 1987
Fact Sheet Number: 122

1. Description of Chemical

Generic Name: 2,6-dichlorobenzonitrile

Common Name: dichlobenil

Trade Names: Casoron®, H-133, Decabane®, 2,6-DBN, Code 133®

EPA Shaughnessy Code: 027401

Chemical Abstracts Service (CAS) Number: 1194-65-6

Year of Initial Registration: 1964

Pesticide Type: Herbicide

Pests Controlled: Broadleaf weeds and grasses (annual and perennial), and aquatic weeds.

Chemical Family: Benzonitrile

U. S. and Foreign Producers: Duphar B.V. (Netherlands); PBI/Gordon Corporation; Shell International Chemical Company, Ltd., London; and Uniroyal Chemical, Div. of Uniroyal, Inc.

2. Use Patterns and Formulations

Application Sites: Terrestrial food and nonfood crops, aquatic nonfood, forestry, commercial and industrial sites.

Types and Methods of Application: Ground or aerial application.

Application Rates: Alfalfa and Ladino Clover 1.4 to 2.0 lb ai/A; 4 to 6 lb ai/A in cranberry bogs, granular mix 3.4 to 6.75 lb ai/A and up to 10 lb ai/A on very weedy bogs; 4 to 6 lb ai/A for

control of annual weeds in bearing, nonbearing, nursery stock, noncitrus, nut crops; 4 to 7 lb ai/A in citrus nurseries; terrestrial nonfood crops 10 to 20 lb ai/A; aquatic weed control 7 to 15 lb. ai/A.

Types of Formulations: 2%, 4%, and 10% active ingredient (ai) granule (G); 50% ai wettable powder (WP); 1.73%, 1.77%, 2.1% and 3.12% ai soluble concentrate/ liquid (SC/L); and 6.75% ai liquid ready to use (RTU).

Usual Carriers: Water.

3. Science Findings

The current data base does not suggest any major toxicological problems. However, there are several toxicology data gaps: acute inhalation, dermal sensitization, primary eye irritation, primary dermal irritation, oncology,reproduction studies and a teratology study in a second species. Dichlobenil has a low acute oral and moderate acute dermal toxicity. Acute inhalation toxicity, primary eye and dermal irritation, and dermal sensitization have not been characterized.

There is a potential toxicity concern regarding the plant and soil metabolite, dichlorobenzamide (BAM). Subchronic ingestion of BAM produces a neuromuscular effect in rats, not observed with the parent compound. This metabolite is readily absorbed and translocated by plants. Because BAM has tentatively been implicated in adverse toxicological symptoms not attributable to dichlobenil per se, we are requiring additional residue data depicting residues of BAM.

Chemical Characteristics: P = Pure
T = Technical

Physical state - (P) Crystalline solid
(T) Powder

Color - (P) White
(T) Pale yellowish

Odor - Characteristic aromatic

Melting point - (P) 145 to 146 °C
(T) 140 to 144 °C

Solubility - (at 20 °C)
(T) 25 ppm in water
(T) 10 g/100 g in methylene chloride
(T) 4 g/100 g in toluene

Vapor pressure - (P) 5.5×10^{-4} mm Hg at 20 °C

Octanol/water partition coefficient - (P) 3.06

Toxicology Characteristics:

Toxicity Category and Value(s) for Each Acute Hazard

Acute Oral Toxicity (rat)	4250 (3510-5150) mg/kg (males and females) Toxicity Category III
Acute Dermal Toxicity (rabbit)	1350 + 158 mg/kg (males) Toxicity Category II

Subchronic Oral Toxicity - Rodent (Rat): Compound-related effects included increased absolute and relative liver and kidney weights at 1000 ppm and above; hepatic degeneration and an absolute neutropenia and leukopenia at 3000 ppm and above; and mortality (5 out of 6) and hepatic necrosis at 10,000 ppm.

No Observed Effect Level (NOEL): 100 ppm (50 mg/kg/day) (rodent)

Lowest Effect Level (LEL): 1000 ppm (500 mg/kg/day) (rodent)

Nonrodent - (beagle dogs): This requirement is satisfied by the 2-year chronic feeding study.

A core-minimum subchronic oral rat study was submitted. Rats (10/sex/dose) were treated with BAM in the diet for 13 weeks at doses of 0, 50, 180, 600, and 2300 ppm. The NOEL (180 ppm [14 mg/kg/day]) and the LEL (600 ppm [49 mg/kg/day]) were based on decreased body weight gain and food efficiency, increased blood urea nitrogen (BUN), and reduced coagulation times. There was also a possible reduction in muscle tone.

Major Routes of Exposure: The major route of exposure is through the skin during mixing and loading.

Chronic Toxicology Results:

Oncogenicity - The oncogenic potential of dichlobenil cannot be determined from the available rat study. A second oncogenicity study (not rat) is needed to determine its oncogenic potential.

Chronic feeding - Nonrodent (beagle dogs): The NOEL and LEL were based on increased absolute and relative liver and thyroid weight in both sexes; leukocytic infiltration and fibrinoid degeneration around the central hepatic veins in both sexes; increases in serum alanine aminotransferase (SGPT) (females) and serum alkaline phosphatase (SAP) (males and females); and liver enzyme glucose-6-phosphatase and glucose-6-phosphatase dehydrogenase (G6Pase and G6PD) activity (males and females).

NOEL = 50 ppm (1.25 mg/kg/day) (nonrodent)

LEL = 350 ppm (8.75 mg/kg/day) (nonrodent)

Teratogenicity - Rodent (Rat):

Maternal Toxic NOEL = 20 mg/kg/ day

Developmental NOEL = 60 mg/kg/day

Mutagenicity: Battery of mutagenicity tests for gene mutation, chromosomal aberration, direct DNA damage and transformation are negative.

Physiological and Behavioral Characteristics:

Translocation - 2,6-Dichlorobenzamide is absorbed by roots and translocated in the plant.

Mechanism of Pesticide Action - It stimulates oxygen utilization and inhibits esterification of phosphorus, resulting in reduced meristemic cell growth and inhibition of germination.

Metabolism and Persistence in Plants and Animals - The available data are inadequate to evaluate the persistence of dichlobenil in plants and animals.

Environmental Characteristics:

Available data are insufficient to fully assess the environmental fate and the potential exposure of humans and nontarget organisms to dichlobenil. Additional data are needed to characterize the potential for dichlobenil to enter ground water.

Ecological Characteristics:

Based on available data, the aquatic use of dichlobenil presents the only major ecological concern. The requirements for acute testing are only partially satisfied. Dichlobenil is slightly toxic to game birds and moderately toxic to fish and aquatic invertebrates.

Toxicity to fish	Moderately toxic to coldwater (LC_{50} = 6.3 mg/L) and warmwater fish (LC_{50} = 5.7 to 8.3 mg/L)
Toxicity to Aquatic Invertebrates	Moderately toxic (EC_{50} = 3.2 ppm
Toxicity to birds	Slightly toxic to pheasants at an LC_{50} = 1500 ppm.

Potential Problems Related to Endangered Species: There are sufficient data to adequately evaluate the hazard to endangered avian and aquatic species.

Tolerance Assessment:

List of Crops and Tolerances - A tolerance at 0.15 part per million (ppm) has been established for the combined negligible residues of dichlobenil and its metabolite 2,6 dichloro-benzoic acid (2,6-DCBA) in or on the raw agricultrual

commodities: almond hulls, apples, avocados, blackberries, blueberries, citrus, cranberries, figs, grapes, mangoes, nuts, pears, raspberries, and stone fruits.

Results of Tolerance Assessment - A Provisional Acceptable Daily Intake (PADI) of 0.00125 mg/kg/day has been established for dichlobenil based on a 2-year dog feeding study and a thousand-fold safety factor. Current tolerances result in a Theoretical Maximal Residue Contribution (TMRC) of 0.000325 mg/kg/day and utilizes 26 percent of the PADI.

4. Summary of Regulatory Position and Rationale

Warning Statements Required on Labels:

Manufacturing use - Do not discharge effluent containing this product into lakes, streams, ponds, estuaries, oceans, or public waters unless this product is specifically identified and addressed in an NPDES permit. Do not discharge effluent containing this product to sewer systems without previously notifying the sewage treatment plant authority. For guidance, contact your State Water Board or Regional Office of the EPA.

End use, aquatic weed control (nonfood) - Do not contaminate untreated water by cleaning of equipment or disposal of wastes. Treatment of weed areas can result in oxygen loss from decomposition of dead weeds. This loss can cause fish suffocation. Therefore, treat only 1/3 of the weed areas at a time and wait 14 days between treatments. Consult your State Environmental Regulatory Agency concerning the need for a permit before applying this product to public waters.

End use, aquatic food (cranberry) - Do not contaminate water by cleaning of equipment or disposal of wastes.

End use, nonaquatic (nongranular) - Do not apply directly to water or wetlands (swamps, bogs, marshes, and potholes). Do not

contaminate water by cleaning of equipment or disposal of wastes.

End use, nonaquatic (granular) - Cover, collect or incorporate granules spilled on the soil surface. Do not apply directly to water or wetlands (swamps, bogs, marshes and potholes). Do not contaminate water by cleaning of equipment or disposal of wastes.

Worker safety label statements

IMPORTANT! Always wash hands, face, and arms with soap and water before smoking, eating, drinking, or toileting. [For nongranular formulations: Before removing gloves, wash them with soap and water.]

Keep all unprotected persons, children, livestock, and pets away from treated area or where there is danger of drift.

Do not rub eyes or mouth with hands. If you feel sick in any way, STOP work and get help right away. See Statement of Practical Treatment.

5. Summary of Major Data Gaps

Data	Due
Product Chemistry	6 to 15 months
Residue Chemistry	
Nature of Residue (Metabolism)	18 Months
Residue Analytical Method	15 Months
Storage Stability	6 Months
Magnitude of Residues For Each Food Use	18 Months
Environmental Fate	
Hydrolysis	9 Months
Photodegradation (water/soil)	9 Months
Metabolism	27 Months
Leaching and Adsorption/ Desorption	12 Months
Dissipation	27 to 50 Months
Accumulation	12 to 50 Months
Toxicology	
Acute Inhalation	9 Months
Primary Eye Irritation	9 Months

Primary Dermal Irritation	9 Months
Dermal Sensitization	9 Months
Chronic Toxicity (rodent)	6 Months
Oncogenicity (mouse)	48 to 50 Months
Oncogenicity (rat)	6 Months
Teratogenicity (non-rodent)	12 Months
Reproduction (rat)	30 Months
General Metabolism	24 Months

Wildlife and Aquatic Organisms

Acute Avian Oral Toxicity	9 Months
Avian Subacute Dietary (waterfowl)	9 Months
Acute Toxicity to Freshwater Invertebrates	9 Months
Fish Early Life Stage	15 Months
Aquatic Invertebrate Life Cycle	15 Months
Aquatic Organism Accumulation	12 Months
Nontarget Area Phytotoxicity	9 Months

6. Contact Person at EPA

Robert J. Taylor
U.S. Environmental Protection Agency
Registration Division (TS-767C)
401 M Street SW.
Washington, D.C. 20460
(703) 557-1800

DISCLAIMER: The information presented in this Pesticide Fact Sheet is for informational purposes only and may not be used to fulfill data requirements for pesticide registration and reregistration.