**United States
Department of
Agriculture**

Forest Service
Pacific Northwest Region

**Healthy Forests Make
A World of Difference**

February 1999

Dicamba

HERBICIDE INFORMATION PROFILE

This information profile is produced by the USDA Forest Service, Pacific Northwest Region, for employees, forest workers, and for the public. The profile provides information on forest and land management uses, environmental and human health effects, and safety precautions for the herbicide dicamba and its formulations. A list of definitions is included in Section VIII of the information profile.

The available information on dicamba has not changed substantially since the previous profile. The U.S. EPA, however, modified the dose/response assessment for dicamba. Both the PNW FEIS and the SERA (1994a) risk assessment used an earlier U.S. EPA dose/response assessment. The changes in U.S. EPA's dose/response assessment are discussed in Section V, and the potential impact of these changes on the interpretation of risk is given in Section VI.

I. BASIC INFORMATION

COMMON NAME: dicamba

CHEMICAL NAME: 3,6-dichloro-o-anisic acid or 3,6-dichloro-2-methoxy benzoic acid

PRODUCT NAMES: Banvel® and Vanquish® products for forestry and non-crop sites.

PESTICIDE CLASSIFICATION: herbicide

REGISTERED USE STATUS: "General Use"

FORMULATIONS: Banvel and Vanquish are currently supplied by BASF (C&P Press 1998, Novartis 1996). The previous profile covered one formulation, Banvel CST, which is not in use.

Dicamba formulations contain one or more inert ingredients. The identity of inert ingredients is usually not listed on the label. The manufacturer revealed the identity of all inerts to the U.S. EPA. Although the Forest Service asked the manufacturer to identify all inert ingredients for public disclosure in this profile, some of the inert ingredients in the formulations were not publicly identified. Nevertheless, hazardous inert ingredients (as defined by U.S. Occupational Health and Safety Administration) were publicly identified. In instances where the manufacturer did not publicly identify inert ingredients, this profile may not fully characterize hazards to human health and the

environment associated with a dicamba formulation.

Manufactured by BASF:

Banvel

Dicamba, as the DMA salt	48.2%
DMA salts of related acids	12.0%
Inert ingredients	39.8%

Vanquish

Dicamba, as the DGA salt	56.8%
DGA salts of related acids	14.2%
Inert ingredients	29.0%

The results of formulation testing reported in this profile apply only to the Banvel and Vanquish products. These products contain only dicamba as an active herbicide ingredient. Other formulated products contain both dicamba and another herbicide. Information in this profile does not address possible effects of these formulated herbicide mixtures.

RESIDUE ASSAY METHODS: 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 is 0.03 ppb with recovery rates ranging from 81.2% to 95%. Other reported limits of detection include 1–10 ppm for soil and 10-20 ppb in plants (SERA 1994b).

II. HERBICIDE USES

REGISTERED FORESTRY, RANGELAND, RIGHT-OF-WAY USES: Dicamba is used in the control of annual and perennial broadleaf weeds, brush, and vines in rangeland and non-cropland areas. Non-cropland areas include fence rows, roadways, rights-of-way, maintenance of wildlife openings, and non-selective forest brush control (including site preparation).

OPERATIONAL DETAILS:

TARGET PLANTS: Dicamba is used to control broadleaf plants, brush, and vines. Dicamba does not injure grasses at recommended application rates.

MODE OF ACTION: Dicamba acts like a naturally occurring plant hormone and causes uncontrolled growth in plants. At sufficiently high levels of exposure, the abnormal growth is so severe that the plant dies.

METHOD OF APPLICATION: Ground or aerial broadcast, soil (band) treatment, basal bark treatment, stump (cut surface) treatment, frill treatment, tree injection, and spot treatment.

USE RATES: Labeled application rates range from 0.25 to 8 lbs./acre. The Forest Service, however, does not use dicamba formulations at the highest application rate. The typical rate used by the Forest Service is 2 lbs./acre in mechanical and backpack foliar applications. For cut surface treatments, the typical application rate is 1.5 lbs./acre.

SPECIAL PRECAUTIONS:

Before using this herbicide, always read all of the information on the product label and material safety data sheet for application and handling instructions and application restrictions.

TIMING OF APPLICATION: Dicamba should generally be applied during periods of active plant growth. Spot and basal bark treatments can be applied when plants are dormant, but should not be done when snow or water prevent application directly to the ground.

DRIFT CONTROL: Do not apply dicamba where it may move down in the soil or be washed along the soil surface to roots of desirable plants. Do not apply when air currents could carry spray to desirable plants. Leave buffer zones between area to be treated and desirable plants. Do not apply near desirable plants on days when the temperature is likely to exceed 85°F. Do not apply from aircraft when desirable plants are growing near the area to be treated. Avoid fine sprays.

IV. ENVIRONMENTAL FATE

SOIL:

RESIDUAL SOIL ACTIVITY: Dicamba may cause damage to plants as a result of its absorption from the soil by plant roots. Half-times of dicamba in soil usually are between 1 and 6 weeks (Cox 1994, Muller and Buser 1997).

ADSORPTION: Dicamba is highly mobile in and poorly adsorbed by most soil types. 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).

PERSISTENCE AND DEGRADATION: Dicamba is moderately persistent in soil. Its reported half-life in soil ranges from 1 to 6 weeks. 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 and Groffman 1997a,b). The slower than expected field dissipation is probably attributable to sorption of dicamba in acidic and highly organic soil horizons.

METABOLITES/DEGRADATION PRODUCTS AND POTENTIAL ENVIRONMENTAL EFFECTS: In soil, dicamba breaks down to very simple substances like carbon dioxide and water. Some intermediates structurally related to dicamba are formed during this process. One of the intermediates, 3,6-dichlorosalicylic acid (3,6-DCSA), is adsorbed to soil much more strongly than is dicamba. Very little information is available on the toxicity of these intermediates (SERA 1994a,b).

WATER:

SOLUBILITY: Dicamba salts used in Banvel and Vanquish formulations are highly soluble in water.

POTENTIAL FOR LEACHING INTO GROUNDWATER: A recent study conducted by the U.S. Geologic Survey (USGS 1998) found dicamba in 0.11%-0.15% of the ground waters surveyed. The maximum level detected was 0.0025 mg/L. 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%). Several additional studies summarized in SERA (1994b) and studies published in the more recent literature (Miller et al. 1995, Ritter et al. 1996) report higher frequencies of occurrence of dicamba in groundwater from agricultural areas.

SURFACE WATERS: Dicamba was detected in 0.32% of stream samples and 0.12% of samples from major aquifers (USGS 1998). The highest level detected was 0.00016 mg/L. In an agricultural area where herbicides are used extensively, dicamba was found in 17%-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 and Elkins 1990). Several additional monitoring studies report low concentrations of dicamba in soil runoff. Usually, however, percolation through soil will predominate over soil runoff (SERA 1994b).

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, then dropped to background levels (<0.001 mg/L) after 37.5 hours. The scientists attributed these residues to drift and direct application of dicamba to water instead of surface runoff (Norris and Montgomery 1975).

AIR:

VOLATILIZATION: Dicamba is relatively volatile, and this process may be a significant factor in the dispersion of dicamba in the environment. In a recent review, Majewski and Capel (1995) cite the occurrence of dicamba, along with several other pesticides, in rain water at sites distant from any known agricultural application. In a small agricultural watershed in Canada, seasonal estimates of the atmospheric deposition of dicamba over a 4-year period ranged from 0.02% to 0.18% of the total amount applied each year (Waite et al. 1995).

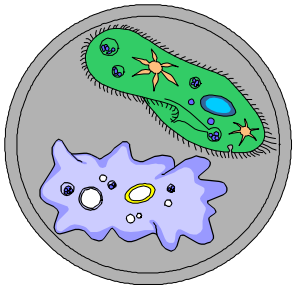
POTENTIAL FOR BY-PRODUCTS FROM BURNING OF TREATED VEGETATION: Brown-and-burn operations may result in the formation of considerable quantities of combustion products. The combustion products of dicamba are not identified (SERA 1994a,b). Because both Banvel and Vanquish contain nitrogen and chlorine molecules, the combustion of these formulations may produce amines, oxides of

nitrogen, and hydrochloric acid (C&P Press 1998).

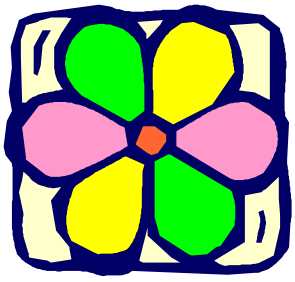
IV. Ecological Effects

Non-target Toxicity:

Soil Microorganisms:
At a level of 10 mg/kg in sandy loam soil, dicamba caused a transient decrease in nitrification after 2 but not 3 weeks of incubation (Tu 1994). The investigator determined that the decrease in nitrification is not substantial and does not suggest the potential for a prolonged impact on microbial activity. In the same study, dicamba did not affect ammonia formation or sulfur oxidation. In a more recent laboratory study, dicamba, at a concentration of 1 mg/kg soil, did not affect urea hydrolysis or nitrification in four soil types (Martens and Bremner 1993). At 50 mg/kg soil, dicamba decreased urea hydrolysis by 6% in one of the four soil types and inhibited nitrification in two of the soils at 7 and 14 but not at 21 days after application.

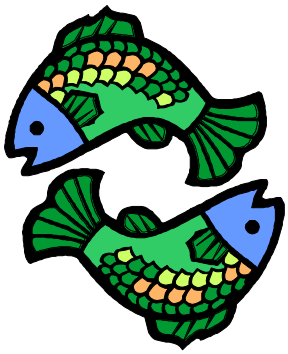


Plants: Dicamba is toxic to many terrestrial broadleaf and conifer species, but is generally less toxic to grasses. Dicamba is relatively toxic to some species of cacti (Crosswhite et al. 1995). These investigators speculate that the formulation of dicamba that they used, which is not specified in the publication, may have contained a surfactant that increased the absorption of dicamba into the cacti.

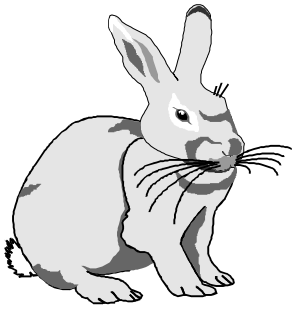


Some aquatic plants are highly sensitive to dicamba, with EC_{50} values for sensitive species between 0.1 and 0.2 ppm. Other plant species are less sensitive, with EC_{50} values greater than 10 ppm (SERA 1994b). A more recent study on the effects of dicamba on aquatic plants (Fairchild et al. 1997) does not alter the risk assessment for aquatic plant species given in SERA (1994a).

Aquatic Animals:
Dicamba was tested for acute toxicity to a variety of aquatic animals. The studies accepted by the U.S. EPA found dicamba acid and DMA salt to be practically nonionic to aquatic invertebrates. Slight toxicity to specific crustaceans was reported in three tests of unknown quality not used by the U.S. EPA. Studies accepted by the U.S. EPA found dicamba acid to be slightly toxic to cold water fish (rainbow trout), and practically nontoxic to warm water fish. Banvel formulations were tested in fish and categorized as practically nontoxic. The U.S. EPA did not require additional testing for Vanquish, based on the low toxicity and bioaccumulation determined in tests using the Banvel formulations.



TERRESTRIAL ANIMALS:



Although the toxicity of dicamba to experimental mammals has been well characterized, little information is available on toxicity to wildlife species. Based on acute toxicity tests dicamba is classified as slightly toxic to experimental mammals. Banvel formulations were less toxic to laboratory mammals than dicamba alone. No tests of formulations for acute toxicity to wildlife mammals have been reported.

The acute toxicity of dicamba to birds is low. Based on acute toxicity tests, dicamba acid is classified as practically nontoxic to duck and quail. In 8-day feeding studies, formulated dicamba acid and salts were practically nontoxic to duck and quail.

Livestock may graze dicamba-treated areas without restriction, unless they are actively producing milk. Meat animals must be removed from treated areas 30 days before slaughter (C&P Press 1998).

No information was found in the published literature regarding the chronic effects of dicamba and its formulations in wildlife species.

THREATENED AND ENDANGERED SPECIES: Dicamba may be a hazard to endangered species if it is used in areas where they live.

V. HEALTH EFFECTS TESTING

Most of the available data on potential human health effects come from laboratory animal studies. These data are evaluated and used to make inferences about potential effects on human health.

For dicamba and formulations containing dicamba as the only active ingredient, the data are from studies conducted by the manufacturer. These studies were submitted to the U.S. EPA to support product registration but are not available to the general public.

ACUTE TOXICITY:

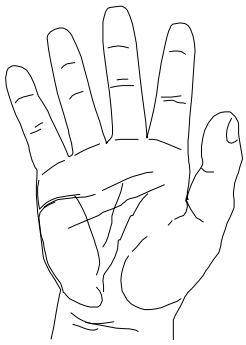
ACUTE ORAL TOXICITY: Based on an acute oral LD_{50} of 2740 mg/kg in rats, the U.S. EPA places dicamba in Category III (Rowland 1998). This category is associated with a code word of *CAUTION* that indicates that the compound may be harmful if swallowed (U.S. EPA 1998). Smaller animals are less sensitive than larger animals to dicamba in acute oral toxicity tests. The lowest reported LD_{50} for dicamba in any species is 566 mg/kg (guinea pigs and rabbits) (SERA 1994b). This LD_{50} of 566 mg/kg would still place dicamba in Category III.

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.

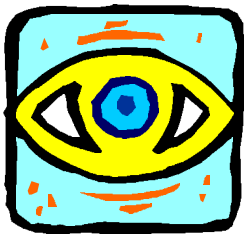
The PNW FEIS and the SERA (1994a) assessments were based on a lower chronic RfD of 0.03 mg/kg/day. Thus, using the U.S. EPA's more recent RfD does not increase the estimates of risk to workers or members of the general public.

ACUTE DERMAL TOXICITY: The toxicity level of dicamba applied directly to skin was greater than 2000 mg/kg in rats. Thus, for acute dermal toxicity, dicamba is classified as Category III with the following verbal interpretation: Harmful if absorbed through skin. Avoid contact with skin, eyes or clothing (Rowland 1998, U.S. EPA 1998).



PRIMARY SKIN IRRITATION SCORE: The U.S. EPA places dicamba into Category II with the following verbal interpretation: WARNING: Causes skin irritation. Do not get on skin or on clothing (Rowland 1998, U.S. EPA 1998).

PRIMARY EYE IRRITATION: The U.S. EPA places dicamba into Category II with the following verbal interpretation: WARNING: Causes substantial but temporary eye injury. Do not get in eyes or on clothing (Rowland 1998, U.S. EPA 1998).



ACUTE INHALATION: The U.S. EPA places dicamba into Category IV and does not require cautionary labeling for inhalation exposure (Rowland 1998, U.S. EPA 1998).

DERMAL SENSITIZATION: The U.S. EPA's reevaluation of dicamba indicates that exposure to dicamba is not associated with skin sensitization (Rowland 1998).

EVALUATION OF SKIN AND EYE IRRITATION: Concentrated solutions of dicamba cause eye irritation. For concentrated solutions of dicamba, the irritation was characterized as severe conjunctival swelling and corneal clouding. Some information suggests that eye irritation may be at least partly attributed to the acidity of some dicamba solutions (SERA 1994b). The extent to which dicamba formulations may cause dermal or ocular irritation during normal use cannot be determined from the available data. The irritant effects of the formulation are likely to depend on its pH and the presence of other adjuvants.

The PNW Region FEIS evaluated the testing as Inadequate for these effects. Since the preparation of the FEIS, the quality of the information is unchanged.

CHRONIC TOXICITY:

The Pacific Northwest Region FEIS risk assessment and the more recent assessments sponsored by the Forest Service (SERA 1994a,b) and the U.S. EPA (Rowland 1998) involve evaluations of data quality and consistency. Please refer to Section X for an explanation of qualitative ratings given in this section.

SYSTEMIC TOXICITY:

REPRODUCTION/DEVELOPMENTAL EFFECTS: This is the most important effect for evaluating longer term exposure to dicamba. The PNW Region FEIS, SERA (1994a,b) and the U.S. EPA (1997b) all use a NOEL of 3 mg/kg/day for reproductive effects from a gavage study (Goldenthal et al. 1978) as the basis for assessing the potential chronic toxicity of dicamba. This NOEL is still listed by the U.S. EPA (1997b) as the basis of an RfD of 0.03 mg/kg/day and confidence in this RfD is categorized as High with the following verbal interpretation:

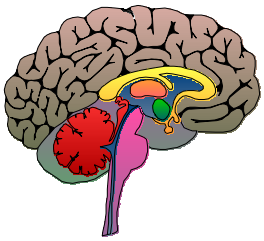
> The critical study is of adequate quality and is given a medium confidence rating. Additional studies are supportive and therefore the data base is given a high confidence rating. High confidence in the RfD follows.

More recently, the U.S. EPA derived a somewhat higher RfD, 0.045 mg/kg/day, that is based on a dietary NOEL of 0.45 mg/kg/day (Miller 1998).

Savitz et al. (1997) recently published an analysis of the association of abnormal pregnancy outcomes with paternal exposure in a group of Canadian farmers. For dicamba, no statistically significant associations were found.

In 1988, the PNW Region FEIS evaluated the testing as Marginal for reproductive and developmental effects. The quality of the database has improved with both the additional studies submitted to U.S. EPA as well as the publication by Savitz et al. (1997).

NERVOUS SYSTEM: Dicamba causes neurological effects in rats, dogs, and hens. The current acute dietary RfD for dicamba of 0.1 mg/kg/day is based on a neurological effect. Recently, Potter et al. (1993) published a study on the inhibition of an enzyme important in neurological function. The details of the study are provided in the appendix to this profile.



In the FEIS, the PNW Region evaluated the testing as Inadequate for nervous system effects. The quality of the data has improved somewhat since the FEIS.

CARCINOGENICITY/MUTAGENICITY: Very little information on the carcinogenic potential of dicamba is available in the recent literature. Mutagenicity studies of dicamba in bacteria, human cells, and whole animals are inconclusive; both positive and negative results are available. In a 2-year rat feeding study, a standard test for carcinogenicity, dicamba was inactive (SERA 1994a,b). Similarly, dicamba does not appear to promote the activity of chemicals that cause cancer (Espandiari et al. 1995, 1996).

Cantor et al. (1992) examined the relationship between exposure to different pesticides, including dicamba, and the development of non-Hodgkin's lymphoma (NHL). The central or best estimates of the relative risks ranged from approximately 1.2 (20% higher than controls) to 3.9 (390%

higher than controls). In no case, however, were the estimates of risk tabulated by Cantor et al. (1992) statistically significant. Nonetheless, in a subgroup of the data, specifically those herbicides marketed before 1965 and used before 1965, Cantor et al. (1992, p. 2450, column 2) state that dicamba use was significantly associated with the risk of NHL but do not provide a tabulated summary of the risk estimates.

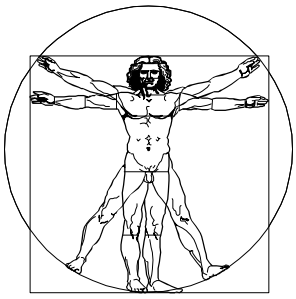
The U.S. EPA recently reviewed the carcinogenicity on dicamba and classified the data on this compound as insufficient to support a quantitative risk assessment (Cogliano 1995).

Consistent with the position of the U.S. EPA, the USDA/FS, both in the 1988 PNW FEIS and in the updated documents (SERA 1994a,b), did not attempt to quantify cancer risk associated with dicamba exposure. The animal data cannot be used because the results are negative (i.e., dicamba did not cause cancer in the bioassays conducted to date). The human data, while raising concern, cannot be used to quantify risk because no reliable dose estimates are available. In addition, the weight of evidence does not clearly support the determination that dicamba is a human carcinogen.

VI. HUMAN HEALTH EFFECTS

FOREST SERVICE EVALUATION OF HUMAN HEALTH RISKS:

The Pacific Northwest Region FEIS and the assessment conducted by SERA (1994a,b) evaluated a range of dicamba health effects data, including some laboratory studies cited in Section V. Both quantitative (numerical) estimates of toxicity, and the quality of data



used to make numerical estimates were evaluated. New information presented in Section V would improve some quality ratings for certain effects and could be used to improve upon some exposure assessments. There are no new studies that substantially alter the identification of hazards. The RfD used in both the PNW FEIS and the SERA (1994a) assessments is more protective (i.e., based on a lower NOEL) than the current U.S. EPA recommendations.

The FEIS Quantitative Risk Assessment predicts the level of human exposure to dicamba for project workers and the public from typical forestry operations and from a large accidental spill. The assessment by SERA (1994a) includes some additional longer-term exposure scenarios for the general public.

The FEIS risk assessment identifies as "Moderate" or "High" any predicted risks from Forest Service operations that exceed U.S. EPA standards. Specific mitigation measures were designed to reduce human exposure from these operations; they are mandatory for projects on PNW Region National Forests. The complete set of risk ratings for the FEIS and the SERA (1994a) assessments are displayed in Section X.

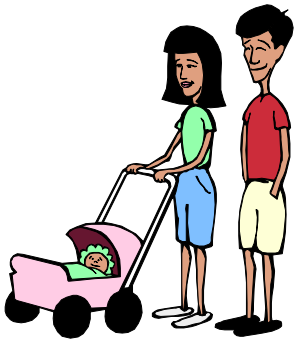
POTENTIAL FOR HEALTH EFFECTS TO THE PUBLIC:

The FEIS and the SERA (1994a) risk assessments indicate that under normal conditions the general public will not be exposed to levels of dicamba that exceed the

RfD. Nonetheless, ample monitoring data show that dicamba can be transported to groundwater and surface water. These monitoring studies, however, do not suggest that typical levels of exposure will approach the RfD. The available information regarding the bioconcentration of dicamba in fish or vegetation does not suggest any plausible hazard associated with the consumption of contaminated fish or vegetation.

MITIGATING MEASURES TO REDUCE IDENTIFIED DICAMBA RISKS TO PUBLIC:

Under extreme conditions involving an accidental spill of dicamba into water or the accidental spraying of an individual, the RfD could be exceeded. If accidental exposures occur, mandatory safety responses (i.e., cleaning the exposed area or the avoidance of further exposure) will help to reduce the possibility of any health consequences. The Forest Service considers the potential for public exposure when designing contact procedures, posting and signing needs in the Herbicide Application Plan. In addition, every effort is made to prevent public contact with accidental spills (emergency spill notification system, restrict public access to the spill site).



PROBABILITY OF A WORKER RECEIVING A DOSE THAT AFFECTS GENERAL HEALTH OR REPRODUCTION:

In the FEIS, the probability of worker exposure to a toxic concentration for general health effects is rated "Low" or "Negligible" for all application methods. The probability of worker exposure to a toxic concentration for reproductive effects is rated "Low" or "Negligible" for aerial and tank truck mixer/loaders and "Moderate" for backpack spray and hack-and-squirt applicators. This assessment is consistent with SERA (1994a): under normal or typical exposure assumptions (i.e., those based on central estimates), workers involved in roadside hydraulic spraying, directed foliar, cut surface, or basal stem treatments do not appear to be at substantial risk. Using the upper limit of the exposure estimates, however, potential reproductive effects would be of concern. At the lower limits of the exposure assumptions, there is no apparent cause for concern.

A balanced conclusion is that dicamba formulations can be applied by roadside hydraulic spraying, directed foliar, cut surface, or basal stem treatments without the risk of significant human health effects as long as workers comply with personal protection standards. If personal protection standards are not practiced by each individual, there is a risk of potential reproductive effects from exposure to dicamba.

MITIGATING MEASURES TO REDUCE IDENTIFIED DICAMBA RISKS TO WORKERS:

In the PNW Region FEIS, Mitigating Measure 13 requires workers applying any herbicide to wear protective clothing. Mitigating Measure 23 requires worker exposure monitoring for all herbicide application projects.

The 1992 Amendment to the ROD requires workers to review this Information Profile before agreeing to apply dicamba herbicides.

The worker may request reassignment without penalty. Additional personal protective equipment will be available at the work site for workers who want to reduce their exposure to the herbicide.

ACUTE TOXICITY (POISONING):

REPORTED EFFECTS: Effects of human exposure to dicamba include muscle cramps, difficult breathing, nausea, vomiting, skin rashes, loss of voice, swollen neck glands, coughing and dizziness.

LONG TERM HUMAN HEALTH EFFECTS:

REPORTED EFFECTS: The only reported effect of long-term exposure to dicamba is the statement by Cantor et al. (1992) that dicamba use is significantly associated with total NHL. As stated above, Cantor et al. (1992) do not provide a tabulated summary of these risk estimates.

POTENTIAL FOR ADVERSE HEALTH EFFECTS FROM INERT INGREDIENTS CONTAINED IN THE FORMULATED PRODUCT:

The manufacturer identified some inert chemicals in dicamba formulations; other inerts were not identified to the public. The identity of all inert ingredients in dicamba formulations were disclosed to the U.S. EPA. The U.S. EPA classifies all inerts into one of four categories, called "Lists". List 1 contains chemicals of known toxic concern. List 2 contains chemicals of suspected toxic concern which are high priority for testing. List 4 contains chemicals of known nontoxic character, generally recognized as safe to humans. All other chemicals are classified on List 3: Inerts of unknown toxicity. U.S. EPA did not find enough information available on the toxic properties of List 3 chemicals to classify them on Lists 1, 2, or 4. All inert ingredients used in Banvel and Vanquish formulations are classified by the U.S. EPA on List 3 or List 4.

HEALTH EFFECTS ASSOCIATED WITH CONTAMINANTS:

Traces of 2,7-dichlorodibenzo-p-dioxin (up to 50 ppb) are formed during production of dicamba. The more toxic dioxin 2,3,7,8-tetrachlorodibenzo-p-dioxin was not found at the 2 ppb detection limit, and is not predicted to be an impurity in dicamba.

DMA salt formulations of dicamba may be contaminated with less than 1 ppm of dimethylnitrosamine. U.S. EPA estimates the risk levels for nitrosamine in these dicamba formulations to be less than 1 in 1,000,000 (EPA 1983).

HEALTH EFFECTS ASSOCIATED WITH OTHER FORMULATIONS:

Some formulations contain dicamba mixed with other herbicides like 2,4-D or atrazine. This profile does not fully describe the potential for health or environmental effects from these formulations containing multiple herbicides. Additional information on properties and potential effects of these formulations will be prepared before they are used in the PNW Region.

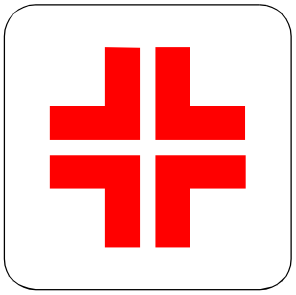
SOCIETAL PERCEPTIONS:

Public opinion about herbicide use in general ranges from a perception that herbicides are completely safe, to a perception that they are very hazardous. A full range of opinion is available in the FEIS. This profile provides

workers and the general public with information that may be useful in assessing the hazards associated with the use of dicamba on PNW Region National Forests.

VII. Safety Precautions

SIGNAL WORDS AND DEFINITIONS: All of the following are taken from product labels and material safety data sheets (C&P Press 1998).



Banvel: WARNING - Causes eye irritation. Harmful if swallowed.

Vanquish: CAUTION - Harmful if swallowed.

SYSTEMIC SIGNS PROTECTIVE PRECAUTIONS FOR WORKERS: Do not get in eyes, on skin, or on clothing. Avoid breathing spray mist. Wash thoroughly after handling.

MEDICAL TREATMENT PROCEDURES: For exposure to the skin, wash with soap and water. For exposure to the eyes, flush with water for 15 minutes and get medical attention. If inhaled, remove the victim to fresh air. Apply artificial respiration if the victim is not breathing; get medical attention. If swallowed and if the victim is conscious, give 1 to 2 glasses of water and induce vomiting by touching the back of the throat with a finger. If the person is unconscious, do not give anything by mouth and do not induce vomiting. Get medical attention. In case of emergency call your local poison control center for advice.

HANDLING, STORAGE AND DISPOSAL: Dicamba is stable under normal storage conditions. Store in the original container in a well-ventilated area separately from fertilizer, animal feeds and food. Do not contaminate water, food, or feeds by storage or disposal. Dispose of waste on site or at an approved waste disposal facility.

EMERGENCY (SPILL) HAZARDS AND PROCEDURES: Dike or contain the spill. Absorb liquid with absorbent material such as sawdust. Place material in a container for later disposal. Observe all local, state, and federal rules for disposal. In case of a large spill, call CHEMTREC at 1-800-424-9300 or BASF at 1-800-832-HELP for advice.

VIII. Definitions

Absorption -- The process by which a chemical passes through the body membranes and enters the bloodstream. The main routes by which toxic agents are absorbed are the gastrointestinal tract, lungs, and skin.

Acute exposure -- A single exposure or multiple exposure occurring within a short time (24 hours or less).

Adjuvant(s) -- Additives to formulations used to enhance the toxic effect of the active ingredient.

Adsorption -- The tendency of one chemical to adhere to another material.

Adverse-Effect Level (AEL) -- Signs of toxicity that must be detected by invasive methods, external monitoring devices, or prolonged systematic observations. Symptoms

that are not accompanied by grossly observable signs of toxicity. In contrast to Frank-effect level.

Assay -- A kind of test (noun); to test (verb).

Bioconcentration Factor -- The concentration of a compound in an aquatic organism divided by the concentration in the ambient water of the organism.

Broadleaf weed -- A nonwoody dicotyledonous plant with wide bladed leaves designated as a pest species in gardens, farms, or forests.

Carcinogen -- A chemical capable of inducing cancer.

Chronic Exposure -- Long-term exposure studies often used to determine the carcinogenic potential of chemicals. These studies are usually performed in rats, mice, or dogs and extend over the average lifetime of the species (for a rat, exposure is 2 years).

Contaminants -- Impurities present in a commercial grade chemical.

Degraded -- Broken down or destroyed.

Dermal -- Pertaining to the skin.

Drift -- That portion of a sprayed chemical that is moved by wind off a target site.

EC_{50} -- A concentration that causes 50% inhibition or reduction. As used in this document, this values refers to a 50% inhibition of growth.

Enzymes -- A biological catalyst; a protein, produced by an organism itself, that enables the splitting (as in digestion) or fusion of other chemicals.

Exposure Assessment -- The process of estimating the extent to which a population will come into contact with a chemical or biological agent.

Formulation -- A commercial preparation of a chemical including any inerts or contaminants.

Gavage -- The placement of a toxic agent directly into the stomach of an animal, using a gastric tube.

Halftime -- For compounds that are eliminated by first-order kinetics, the time required for the concentration of the chemical to decrease by one-half.

Herbicide -- A chemical used to control, suppress, or kill plants, or to severely interrupt their normal growth processes.

Hydrolysis -- Decomposition or alteration of a chemical substance by water.

Inerts -- Adjuvants or additives in commercial formulations of glyphosate that are not readily active with the other components of the mixture.

Invertebrate -- An animal that does not have a spine (backbone).

Irritant Effect -- A reversible effect, compared with a corrosive effect.

Lowest-Observed-Adverse-Effect Level (LOAEL) -- The lowest dose of a chemical in a study, or group of studies, that produces statistically or biologically significant increases in frequency or severity of adverse effects between the exposed population and its appropriate control.

Malignant -- Cancerous.

Margin of safety (MOS) -- The ratio between an effect or no effect level in an animal and the estimated human dose.

Metabolite -- A compound formed as a result of the metabolism or biochemical change of another compound.

mg/kg -- A common way of expressing dose: milligram of a toxic agent per kilogram of body weight.

Microorganisms -- A generic term for all organisms consisting only of a single cell, such as bacteria, viruses, and fungi.

Mutagenicity -- The ability to cause genetic damage (that is damage to DNA or RNA). A mutagen is substance that causes mutations. A mutation is change in the genetic material in a body cell. Mutations can lead to birth defects, miscarriages, or cancer.

Non-target -- Any plant or animal that a treatment inadvertently or unavoidably harms.

No-Observed-Adverse-Effect Level (NOAEL) -- The dose of a chemical at which no statistically or biologically significant increases in frequency or severity of adverse effects were observed between the exposed population and its appropriate control. Effects may be produced at this dose, but they are not considered to be adverse.

No-Observed-Effect Level (NOEL) -- The dose of a chemical at no treatment-related effects were observed.

Ocular -- Pertaining to the eye.

Perennial -- A plant species having a lifespan of more than 2 years.

pH -- The negative log of the hydrogen ion concentration. A high pH (>7) is alkaline or basic and a low pH (<7) is acidic.

ppb -- An abbreviation for *parts per billion*. Equivalent to µg/L for concentrations in water and to µg/kg for concentrations in soil or other non-aqueous media.

ppm -- An abbreviation for *parts per million*. Equivalent to mg/L for concentrations in water and to mg/kg for concentrations in soil or other non-aqueous media.

Reproductive Effects -- Adverse effects on the reproductive system that may result from exposure to a chemical or biological agent. The toxicity of the agents may be directed to the reproductive organs or the related endocrine system. The manifestations of these effects may be noted as alterations in sexual behavior, fertility, pregnancy outcomes, or modifications in other functions dependent on the integrity of this system.

RfD -- A daily dose which is not anticipated to cause any adverse effects in a human population over a lifetime of exposure. These values are derived by the U.S. EPA.

Systemic Toxicity -- Effects that require absorption and distribution of a toxic agent to a site distant from its entry point at which point effects are produced. Systemic effects are the obverse of local effects.

Toxicity -- The inherent ability of an agent to affect living organisms adversely.

Uncertainty Factor (UF) -- A factor used in operationally deriving the RfD and similar values from experimental data. UFs are intended to account or (1) the variation in sensitivity among members of the human population; (2) the uncertainty in extrapolating animal data to the case of humans; (3) the uncertainty in extrapolating from data obtained in a study that is less than lifetime exposure; and (4) the uncertainty in using LOAEL data rather than NOAEL data. Usually each of these factors is set equal to 10.

Volatile -- Referring to compounds or substances that have a tendency to vaporize. A material that will evaporate quickly.

IX. Information Sources

For general information on herbicide use by the Forest Service, refer to the PNW Region Treatment Methods Profile for Herbicides.

The principal sources of information and findings in this profile are the PNW Region FEIS (Final Environmental Impact Statement) for Managing Competing and Unwanted Vegetation (USDA/FS 1988) and a more recent risk assessment on Vanquish, a dicamba formulation, prepared for the Forest Service (SERA 1994a,b).

Update literature searches covering the period from 1992 to 1998 were conducted using four databases: AGRICOLA, Life Sciences Collection, CAB Abstracts, and Medline. In addition, recent assessments of dicamba by the U.S. EPA were obtained from published documents (U.S. EPA 1997a,b), by direct contact with EPA personnel (Ottley 1998; Miller 1998) and through the Freedom of Information Act (Rowland 1998). Various parties interested in the use of dicamba in the PNW Region were made aware of the update process and invited to submit any information that they considered relevant. Only one group, the Northwest Coalition for Alternatives to Pesticides (Grier 1998) submitted new information.

Beasley VR; Arnold EK; Lovell RA. 1991. 2,4-D toxicosis: A pilot study of 2,4-D induced and dicamba-induced myotonia in experimental dogs. Vet. Hum. Toxicol. 33(5): 435-440.

Cantor KP; Blair A; Everett G; Givson R; Burmeister LF; Brown LM; Schuman L; Dick FR. 1992. Pesticides and other agricultural risk factors for non-Hodgkin's lymphoma among men in Iowa and Minnesota. Cancer Research. 52:2447-2455.

Cogliano J. 1995. U.S. EPA. (202)260-2575. Personal communication to P. Durkin, SERA, Inc., July 27, 1995.

Cox C. 1994. Dicamba.. Journal of Pesticide Reform. 14 (1):30-35.

C&P Press (Chemical and Pharmaceutical Press). 1998. EPR II for Windows. Electronic Pesticide Reference.

Crosswhite FS; Feldman WR; Minch EW. 1995. Impact of herbicides on cacti.. Desert Plants. 11 (4):9-31.

Espandiari P; Thomas VA; Glauert HP; O'Brien M; Noonan D; Robertson LW. 1995. The herbicide dicamba (2-methoxy-3,6-dichlorobenzoic acid) is a peroxisome proliferator in rats.. Fundamental and Applied Toxicology. 26 (1):85-90.

Espandiari P; Glauert HP; Lee EY; Robertson LW. 1996. Lack of promoting activity by Dicamba in two stage hepatocarcinogenesis (Meeting abstract). Proc Annu Meet Am Assoc Cancer Res. 37 :A1114.

ExoToxNet 1995. Dicamba. Pesticide Information Notebook. Pesticide Management Education Program, Cornell University, Ithaca, NY. http://www.uoguelph.ca/GTI/urbanpst/dicamba.htm, last Updated December 6, 1995.

Fairchild JF; Ruessler DS; Haverland PS; Carlson AR. 1997. Comparative sensitivity of *Selenastrum capricornutum* and *Lemna minor* to sixteen herbicides. Arch. Environ. Contam. Toxicol.. 32 (4):353-357.

Goldenthal EI; Jessup DC; Rodwoll DE. 1978. Teratology study in rabbits: IRDC No. 163-436. MRID 00028236.

Grier N. 1998. Executive Director, Northwest Coalition for Alternatives to Pesticides. Letter to Leslie Rubin, USDA-APHIS.

Grover R; Waite DT; Cessna AJ; Nicholaichuk W; Irvin DG. 1997. Magnitude and persistence of herbicide residues in farm dugouts and ponds in the Canadian prairies. Environmental toxicology and chemistry. 16 (4): 638-643.

Harris SA; Solomon KR. 1992. Human exposure to 2,4-D following controlled activities on recently sprayed truf. J. Environ. Sci. Health. B27(1): 9-22.

Hoffman DJ, Albers PH. 1984. Evaluation of Potential Embryotoxicity and Teratogenicity of 42 Herbicides, Insecticides, and Petroleum Contaminants to Mallard Eggs. Archives of Environmental Contamination and Toxicology 13:15-27.

Holovska K; Lenartova V; Rosival I; Kicinkova M; Majerciakova A; Legath J. 1998. Antioxidant and detoxifying enzymes in the liver and kidney of pheasants after intoxication by herbicides MCPA and ANITEN I.. J Biochem Mol Toxicol. 12 (4):235-44.

Hrelia P; Vigagni F; Maffei F; Morotti M; Colacci A; Perocco P; Grilli S; Cantelli-Forti G. 1994. Genetic safety evaluation of pesticides in different short-term tests.. Mutat Res. 321 (4):219-28.

Majewski MS; Capel PD. 1995. Pesticides in the Atmosphere: Distribution, Trends, and Governing Factors. Ann Arbor Press, Inc. Chelsea, Michigan. Copy courtesy of Grier (1998).

Martens DA; Bremner JM. 1993. Influence of herbicides on transformations of urea nitrogen in soil. J. Environ. Sci. Health Part B. Pestic. Food Contam. Agric. Wastes. 28(4): 377-395.

Mayer J; Elkins N. 1990. Potential for Agricultural Pesticide Runoff to a Puget Sound Estuary: Padilla Bay, Washington. Bull. Environ. Contam. Toxicol. 45:215-222.

Miller J. 1998. Office of Pesticide Programs. U.S. EPA. E-mail to P. Durkin concerning RfD for dicamba. Nov. 18, 1998.

Miller JJ; Foroud N; Hill BD; Lindwall CW. 1995. Herbicides in surface runoff and groundwater under surface irrigation in southern Alberta. Canadian Journal of Soil Science. 75 (1): 145-148.

Muller MD; Buser HR. 1997. Conversion reactions of various phenoxyalkanoic acid herbicides in soil. 1. Enantiomertization and enantioselective degradation of the chiral 2-phenoxypropionic acid herbicides. Environmental Science and Technology. 31 (7): 1953-1959.

Nishioka MG; Burkholder HM; Brinkman MC; Gordon SM; Lewis RG. 1996. Measuring transport of lawn-applied herbicide acids from turf to home:

correlation of dislodgeable 2,4-D turf residues with carpet dust and carpet surface residues.. Environmental Science & Technology. 30 (11):3313-3320.

Norris, LA; Montgomery, MM. 1975. Dicamba residues in streams after forest spraying. Bull. Environ. Contam. Toxicol. 13(1): 1-8.

Novartis. 1996. Sandoz sells a portion of its corn herbicide business to BASF. http://www.novartis.com/media/releases/novatris96.html.

Ottley M. 1998. EPA/OPP RfD on Dicamba. E-mail to Leslie Rubin, USDA, dated 12/4/98.

Potter WT; Garry VF; Kelly JT; Tarone R; Griffith J; Nelson RL. 1993. Radiometric assay of red cell and plasma cholinesterase in pesticide appliers form Minnesota. Toxicol. Appl. Pharmacol. 119(1): 150-155.

Ritter WF; Chirnside AEM; Scarborough RW. 1996. Leaching of dicamba in a coastal plain soil. Journal of Environmental Science and Health. Part A. 31 (3): 505-517.

Rowland J. 1998. Dicamba - Report of the Hazard Identification Assessment Review Committee. EPA internal memorandum dated January 15, 1998. FOIA copy to Patrick Durkin, SERA Inc., courtesy of Janet Bressant, Office of Pesticide Programs, U.S. EPA.

Savitz DA; Arbuckle T; Kaczor D; Curtis KM. 1997. Male pesticide exposure and pregnancy outcome.. American Journal of Epidemiology. 146 (12):1025-1036.

SERA (Syracuse Environmental Research Associates, Inc.). 1994a. Vanquish Risk Assessment. SERA TR 95-22-02f, dated October 16, 1995. USDA/Forest Service Contract 43-3187-5-0787.

SERA (Syracuse Environmental Research Associates, Inc.). 1994b. Vanquish Chemical Background Statement. TR 95-22-01f, dated October 18, 1995. USDA/ Forest Service Contract 43-3187-5-0787.

Smith SH; O'Loughlin CK; Salamon CM; 1981. Teratology study in albino rats with technical dicamba. Toxigenetics Study No. 450-0460. MRID 00084024. Summarized in SERA 1994a.

Tu CM. 1994. Effects of herbicides and fumigants on microbial activities in soil. Bull. Environ. Contam. Toxicol. 53(1): 12-17.

USDA/FS (U.S. Department of Agriculture/Forest Service). 1988. Pacific Northwest Region, Forest Service, U.S. Department of Agriculture. 1988. Final Environmental Impact Statement for Managing Competing and Unwanted Vegetation.

U.S. EPA (U.S. Environmental Protection Agency). 1983. Office of Pesticide Programs. Pesticide Fact Sheet Number 8: Dicamba. 6 pp.

U.S. EPA (U.S. Environmental Protection Agency). 1988. Office of Drinking Water. 1988. Dicamba Health Advisory. 16 pp.

U.S. EPA (U.S. Environmental Protection Agency). 1997a. Notice of Filing of Pesticide Petitions. Federal Register. 62(228): 63164-63168.

U.S. EPA (U.S. Environmental Protection

Agency). 1997b. Dicamba. IRIS - Integrated Risk Information System. http://www.epa.gov/ngispgm3/iris/subst/0223.htm. File last updated 04/01/97. Oral RfD Assessment last revised 07/01/92.

U.S. EPA (U.S. Environmental Protection Agency). 1998. Label Review Manual. Chapter 8: Precautionary Labeling. Revised August 10, 1998. http://www.epa.gov/oppfead1/labeling/lrm/chap-08.htm.

USGS (U.S. Geological Survey). 1998. Data on Pesticides in Surface and Ground Water of the United States., Results of the National Water Quality Assessment Program (NAWQA). Revised Oct. 23, 1998. http://wwwdwatcm.wr.usgs.gov/cppt/pns_data/data.html

Voos G; Groffman PM. 1997a. Dissipation of 2,4-D and dicamba in a heterogeneous landscape. Applied soil ecology : a section of Agriculture, Ecosystems &. 5 (2):181-187.

Voos G; Groffman PM. 1997b. Relationships between microbial biomass and dissipation of 2,4-D and dicamba in soil.. Biology and Fertility of Soils. 24 (1):106-110.

Waite DT; Grover R; Westcott ND; Irvine DG; Kerr LA; Sommerstad H. 1995. Atmospheric deposition of pesticides in a small southern Saskatchewan watershed.. Environmental Toxicology and Chemistry. 14 (7):1171-1175.

Zhao HT; Jaynes WF; Vance GF. 1996 Sorption of the ionizable organic compound, dicamba (3,6-dichloro-2-methoxy benzoic acid), by organo-clays. Chemosphere. 33 (10): 2089-2100.

X. Toxicity and Risk Categories

ESTIMATES OF HEALTH RISKS TO THE PUBLIC AND TO WORKERS FROM FOREST SERVICE OPERATIONS

The FEIS 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 effect (No Observed Effects Level/NOEL). This level of exposure is referred to as the Margin of Safety or Margin of Exposure approach. The SERA (1994a) risk assessment used a conceptually similar approach in which the estimated level of exposure is divided by some estimate of acceptable exposure. Both the FEIS and the SERA (1994a) assessment also express risk qualitatively. In the FEIS, the risk is ranked from "Negligible" to "High" based on the margin between the expected human dose and the highest NOEL "no effect" dose. A "High" risk rating means that the highest NOEL dose is not more than 10 times larger than predicted human dose under the specified conditions. A "Moderate" risk rating means that the highest NOEL dose is between 10 and 100 times larger than the expected human dose.

As illustrated in the following tables, the qualitative expression of risk for both workers and the general public is reasonably consistent between the FEIS and the updated SERA (1994a) risk assessments. The PNW Region determined that no new information summarized in this profile or in SERA (1994a) would change the public or worker mitigations in the 1988 FEIS, which were based on potential human health risks.

Estimated Health Risks To Project Workers[1].			
Scenario	Risk Category		
	Typical	Lower	Upper
Directed ground[2]	Moderate/ *Low*	Low/ *Negligible*	High
Broadcast ground spray[3]	Low	Negligible	Moderate/ *High*
Aerial	Low	Low	High

[1] From PNW FEIS and SERA 1994a. Where risk classification differ in the two assessments, the classification from SERA is presented in italics.
[2] Backpack, cut surface, and streamline
[3] Boomspray

Estimated Health Risks To The Public[1].			
Scenario	Risk Category		
	Typical	Lower	Upper
Accidental Spray[2]	Negligible/ *Moderate*	Negligible/ *Low*	Low/ *Moderate*
Dermal, vegetation	Negligible	Negligible	Negligible
Contamin-ated fruit [3]	Negligible	Negligible	Low
Contamin-ated water [3]	Negligible	Negligible	Low/ *Negligible*
Contamin-ated fish [3]	Negligible	Negligible	Negligible

[1] From PNW FEIS and SERA 1994a. Where risk classification differ in the two assessments, the classification from SERA is presented in italics.
[2] PNW is based on spray drift. SERA 1994a assessment is based on direct spray.
[3] PNW is based on short-term exposures. SERA 1994a assessment is based on longer-term exposures.

ECOTOXICOLOGIC CATEGORIES

Mammalian (Acute Oral):	
mg/kg	**Risk Category**
<10	very highly toxic
10-50	highly toxic
51-500	moderately toxic
501-2000	slightly toxic
>2000	practically non toxic

Avian (Acute Oral):	
mg/kg	**Risk Category**
<10	very highly toxic
10-50	highly toxic
51-500	moderately toxic
501-2000	slightly toxic
>2000	practically non toxic

Avian (Dietary):	
mg/kg	**Risk Category**
<50	very highly toxic
50-500	highly toxic
501-1000	moderately toxic
1001-5000	slightly toxic
>5000	practically non toxic

Aquatic:	
ppm	**Risk Category**
<0.1	very highly toxic
0.1-1	highly toxic
>1-10	moderately toxic
>10-100	slightly toxic
>100	practically non toxic

TABLES OF CATEGORIES OF TOXICITY

Human Hazards				
		Route of Administration		
Risk Category	**Signal Word**	**Oral (mg/kg)**	**Dermal (mg/kg)**	**Inhalation (mg/kg)**
I	**DANGER -- Poison**	0-50	0-200	0-0.2
II	**WARNING**	>50-500	>200-2000	>0.2-2.0
III	**CAUTION**	>500-5000	>2000-20,000	>2.0-20
IV	**NONE**	>5000	>20,000	>20

	Hazard	
Category	**Eye Irritation**	**Skin Irritation**
I	Corrosive: corneal opacity not reversible within 7 days	corrosive
II	corneal opacity reversible within 7 days; irritation persisting for 7 days	severe irritation at 72 hours
III	no corneal opacity; irritation reversible within 7 days	moderate irritation at 72 hours
IV	no irritation	mild or slight irritation at 72 hours

Category of Quality of Health Effects Data	
Inadequate:	Inadequate information available for evaluating toxicity. There were too few studies of sufficient quality to yield useful or reliable information.
Marginal-Inadequate:	Some useful information exists for evaluating toxicity. There were studies of marginal quality that provided useful information, but studies were inconsistent and some contained flaws. It is likely that new studies would change estimates of health effects.
Marginal:	Marginal but useful information available for evaluating toxicity. There were studies of adequate quality, and results did not vary greatly, but more information would increase reliability. Although new studies may change estimates of health effects, the results are considered moderately reliable.
Adequate:	Adequate information is available. Studies are of sufficient quality and quantity that estimates of human health are considered reliable. New studies are unlikely to change estimates of health effects..

APPENDIX TO DICAMBA PROFILE

This appendix contains some additional details of more recent studies as well as details of some of the more important older studies used in the FEIS or SERA (1994a,b). This technical discussion may be of limited interest to most readers of this profile. Those interested in a fuller discussion of the potential hazards associated with the use of dicamba should consult the PNW FEIS, SERA (1994a,b), or other information sources given in Section IX..

Much of the newer literature on dicamba relates to environmental fate and transport. These studies do not change the information in the profile, although they would be used in any update/revision of a full risk assessment.

Since the previous information profile, very little new information was published regarding toxicity to humans, experimental mammals, or wildlife species. Two new reviews were published (Cox 1994, ExoToxNet 1995), and information from these reviews were considered in the update of this profile. A recent study by Savitz et al. (1997), reporting potential reproductive effects in humans, also was reviewed in detail. Other new studies on the lack of promoting activity (Espandiari et al. 1996) or mutagenic activity (Hrelia et al. 1994) are consistent with previous data and do not change the assessment of risk. The information on peroxisomal proliferation is rats (Espandiari et al. 1995) is consistent with the data reported in SERA (1994a,b). Crosswhite et al. (1995) studied the effects of dicamba on cacti, and the study is useful in its elaboration about the effects of dicamba on non-target terrestrial plants. A more recent study on a single cell freshwater algae (*Selenastrum capricornutum*) and a multicellular aquatic plant (*Lemna minor*) indicates that these species are relatively insensitive to dicamba with EC50 values of approximately 30 mg/L (Fairchild et al. 1997). Thus, this new information does not alter the risk assessment for aquatic plant species given in SERA (1994a). Similarly, the transient effects of dicamba on soil bacteria (Tu 1994) and the impact of bacterial populations on the fate of dicamba (Voos and Groffman 1997a,b) do not have a substantial impact on the assessment of potential ecological effects.

Bird eggs may be sensitive to direct applications of dicamba (Hoffman and Albers 1984). In this study, mallard eggs were immersed in aqueous emulsions of dicamba for 30 seconds and observed to hatch and post-hatch. The precise concentrations of dicamba used are not specified in the study, and the LC50 is reported as ">200 times the field level of application" (also not specified). The effects observed in the surviving birds include reduced growth and stunted eye development; however, the study does not provide details about the incidence of these malformations or the magnitude of the growth reductions. Other than a recent study on the effect of a mixture containing dicamba on the induction of certain enzymes in pheasants (Holovska et al. 1998), no new information regarding the toxicity of dicamba to birds was found in the literature.

Neurological effects are endpoints of concern in the assessment of dicamba. In a 3-generation reproduction study (Smith et al. 1981), ataxia, salivation, and decreased motor activity were observed in rats given a dose of 400 mg/kg/day dicamba. These signs of toxicity are consistent with AChE inhibition. The NOEL for these effects was 160 mg/kg/day. In hens, some nerve damage was noted for 316 mg/kg/day, the highest dose tested (U.S. EPA 1988). In a clinical study using a only one dog, which had been used in toxicity studies of 2,4-D but was given 1 month to recover, a single oral dose of 87 mg/kg dicamba induced clinical signs of toxicity—ataxia, falling, and lethargy—as well as abnormal electromyographic responses. A single dose of 1 mg/kg dicamba given to the same dog, again after a 1-month recovery period, induced no overt signs of toxicity but did cause abnormal electromyographic responses (Beasley et al. 1991).

In addition to these studies on whole animals, Potter et al. (1993) observed that 3,6-dichloro-2-methoxy benzoic acid, the major isomer in dicamba, causes inhibition of both plasma and red blood cell [RBC] cholinesterase, in vitro. Although these investigators indicate that the assays used 3,6-dichloro-2-methoxy benzoic acid, the reported methods indicate that the test material was commercial grade Banvel (the dimethylamine salt), provided by Sandoz Agro, Inc. As part of

the same study, these investigators report an increase in the incidence of AChE inhibition in farm workers using herbicides. The effects in the farm workers, however, were not associated specifically with dicamba exposure. Studies regarding *in vivo* AChE inhibition in experimental mammals after exposure to dicamba were not located in the available literature.

Espandiari et al. (1995) found that dicamba causes certain enzyme changes in rats (i.e., liver peroxisome proliferation), which may be suggestive of potential carcinogenic activity, particularly in promoting the effect of primary cancer causing agents. Nonetheless, in a standard assay for this type of promoting activity, dicamba was inactive (Espandiari et al. 1996).

One of the exposure pathways considered in both risk assessments is uptake from contaminated vegetation. SERA (1994a) used the general assumption, adopted from Harris and Solomon (1992), that the dislodgeable residue would equal 10% of the nominal application rate. A more recent study by Nishioka et al. (1996) suggests much smaller values of 0.1%-0.2% for both dicamba and 2,4-D. Although there may be justification for using these more recent estimates, with the consequent reduction in levels of absorbed dose and risk, this approach was not adopted because the methods used to measure dislodgeable residues in the two studies are substantially different and it is unclear which method is most appropriate for most potential human exposures. Consequently, the most conservative estimate of 10% is maintained.