DOE/EIS - 0097-F

Final Environmental Impact Statement

BONNEVILLE POWER ADMINISTRATION

TRANSMISSION FACILITIES VEGETATION MANAGEMENT PROGRAM

U.S. Department of Energy

August 1983

Appendices

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and Emergency Preparedness

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INTRODUCTION: TOXICOLOGY OVERVIEW

Background Statements concerning the chemical and physical properties, effects on plants and animals, and the environmental fate of herbicides used in BPA's proposed vegetation management program are contained in Appendix A. These statements were compiled using current scientific literature and reference books on herbicides. Background statements have been prepared for the chemical active ingredients comprising the commercial herbicide formulations used by BPA.

In Appendix A the statements are arranged in alphabetical order by the common names of the active ingredients. A number of the commercial herbicide formulations contain several different active ingredients (see Table 3-2). Sodium chlorate and sodium metaborate, active ingredients in Oxy Ureabor, are treated together in one background statement; 2,4-D and 2,4-DP also are treated together in one statement because of their chemical and toxicological similarities. All of the other herbicides are the subject of individual Background Statements. Background Statements for two drift-control additives (Norbak and Lo-Drift) and for diesel oil (an herbicide carrier) are also contained in Appendix A. These statements are the foundation for the General Hazard Assessment of Herbicide Use (see Chapter 7 section on Public Health).

The Background Statements are presented according to the outline shown in Table A-1. In the following paragraphs, the content of the statements will be previewed. Methods and techniques used to develop test data will be described briefly, and the relevance of the data to herbicide hazard assessment will be indicated. Persons desiring more detailed information are referred to books such as: Pesticides Studied in Man (Hayes 1982), Toxicology: The Basic Science of Poisons (Doull, Klaassen and Amdur 1980); Herbicides: Chemistry, Degradation, and Mode of Action (Kearney and Kaufman 1976); and Herbicides: Physiology, Biochemistry, Ecology (Audus 1976). A glossary of technical terms follows the Background Statements.

Table A-1. Outline for Herbicide Background Statements

Chemical Identification

Action in Vegetation

Utilization by BPA

Chemical Fate and Distribution in the Environment

- A. Soil
- B. Water
- C. Air

Chemical Toxicology in Animals and Humans

- A. Acute effects
- B. Chronic effects

Potential Impact on Nontarget Organisms

- A. Vegetation
- B. Fish
- C. Wildlife
- D. Livestock

Hazard Assessment for Current Use Practices

Chemical Identification

The herbicides which are the active ingredients of the various commercial formulations used in vegetation management are specific chemicals, known to most people by their common names. The Environmental Protection Agency assigns each formulation a registration number and approves each for certain uses as specified on the herbicide label. The systematic (chemical) name of the herbicide must be listed on the approved label. Inquiries about the fate and effects of herbicides can be most easily addressed when a complete chemical identification is known.

Physical properties of the chemicals are briefly listed. The solubility of an herbicide in water and organic solvents, and the vapor pressure of the chemical are two particularly important properties which strongly influence the substance's environmental behavior.

The herbicides proposed for use by BPA are listed in Table A-2. Three inorganic salts and 13 organic chemicals are used in present vegetation management programs. The chemical and physical properties of herbicides within each group of chemicals (inorganic salts, acids, triazines, ureas) are similar to each other. Decisions concerning the use of particular substances may be based in part upon the toxic effects and the environmental behavior of the chemicals within a particular group. For example, each of the inorganic salts are highly water soluble. Therefore, they may be too easily leached to be effective in sandy, porous soils which receive excessive rainfall. Similarly, each of the organic acids are strongly adsorbed by soil organic matter. Thus, they tend to be most concentrated in the upper layers of soils that are rich in organic matter.

Action in Vegetation

The killing action of herbicides on plants (phytotoxicity) is the result of complex chemical and physical processes which determine the amount of herbicide reaching sensitive sites within a plant. In order to develop an understanding of the mode of action of herbicides, processes including absorption, translocation, chemical degradation (biotransformation), and biochemical effects are studied. In spite of intensive scientific investigation since the mid-1940s, the precise mechanisms of action of many herbicides remain unknown. Studies of mechanisms of action have contributed important knowledge about how herbicides affect plants. This knowledge guides the development, formulation, and use of herbicides in vegetation management.

Table A-2. Chemical Classification and Primary Phytotoxic Action of Herbicides Proposed for Use by BPA

Chemical Classification	Herbicide[1] Active Ingredient	Phytotoxic Action[2]
Inorganic Salt	Ammonium sulfamate	Unspecific metabolic effects
Organic Acid		
Amino	Glyphosate	Inhibition of plant metabolism
Benzoic	Dicamba	Prolonged abnormal plant growth
Phenoxy	2,4-D (amine, ester)	Prolonged abnormal plant growth
	2,4-DP	Prolonged abnormal plant growth
Nitrile	Dichlobenil	Inhibition of germination and seedling growth
Triazine	Atrazine	Inhibition of photosynthesis
	Prometone	Inhibition of photosynthesis
	Simazine	Inhibition of photosynthesis
Uracil	Bromacil	Inhibition of photosynthesis
Urea	Diuron	Inhibition of photosynthesis
	Monuron	Inhibition of photosynthesis
	Tebuthiuron	Inhibition of photosynthesis

FOOTNOTES:

[1] Common names according to the Herbicide Handbook (Weed Science Society of America 1979).

[2] For details, see Mode of Action of Herbicides (Ashton and Crafts 1973).

Selectivity is a particularly important property of herbicides. When one plant species is more susceptible to a particular chemical than other species, the chemical is said to be selective for that species. For example, 2,4-D is a selective herbicide that is effective in the control of annual and perennial broadleaf weeds at low rates (1/2 to 2 pounds/acre) and in the control of woody plants and brush at high rates (to 8 pounds/acre). At these rates, grasses are tolerant. This selectivity is important for the maintenance of grasses to avoid soil erosion on treated rights-of-way.

The primary phytotoxic actions of herbicides used by BPA are listed in Table A-2. For the chemicals listed, it is noteworthy that only five general types of effects are listed. Furthermore, the processes that are sensitive to the herbicides are processes that are unique to plants. This factor is important in assessing the degree of hazard presented by these herbicides to animals and humans.

To be effective, herbicides must penetrate plants and be absorbed by plant cells. Herbicides applied to soil may be taken up and transported with the water transpiration stream from roots to aerial parts of plants. Foliar-applied materials may be translocated with plant nutrients or photosynthates in the phloem or xylem (i.e., tissues important in transporting water, nutrients, and photosynthates within plants).

Herbicides that are readily absorbed from soil include bromacil, dichlobenil, the ureas, the triazines, sodium chlorate, sodium metaborate, and picloram. Foliar sprays that are translocated include the inorganic salts and the organic acids. These categories are not mutually exclusive. For example, picloram is phytotoxic both as a foliar spray and as a soil-applied herbicide on noncroplands.

Herbicides are metabolized (degraded) in plants and relative rates of degradation can be an important selectivity factor. Usually the degradation products are more water soluble than the parent compound and are less phytotoxic. The ultimate degradation of herbicides is accomplished by soil microflora. All of the chemicals used by BPA in vegetation management are extensively metabolized to nontoxic products. Persons desiring detailed discussion of herbicide metabolism are referred to the comprehensive review, Herbicides: Chemistry, Degradation and Mode of Action, edited by Kearney and Kaufman (1976).

Utilization by BPA

Chemical hazards to the public or to the environment depend both on toxicity of a chemical and on the level of exposure to living things. This section of the Background Statements describes types of sites, methods of application, and application rates for each herbicide as used by BPA. For example, some herbicides are used only at substations for weed control; others are used in broadcast aerial applications on rights-of-way. BPA's specific pattern of use of herbicides therefore determines the probability of exposing the public or various environmental components (e.g., streams, wildlife) to a given amount of herbicide. The pattern of use includes measures taken to minimize exposure (e.g., buffers, drift-control) as well as application methods.

Chemical Fate and Distribution in the Environment

This section of the Background Statements describes the chemical fate and distribution of the various herbicides in soil, water, and air. The chemical fate and distribution of herbicides in the environment will determine both the effectiveness of the agents in vegetation management as well as the magnitude of unintended and unavoidable exposures.

In discussions of environmental residues and persistence of herbicides, it is critical that phytotoxic levels and chemically-detectable levels be carefully distinguished. Of first concern is selection of an application method which will achieve the goals of the vegetation management program. For this purpose, rates of herbicide application are most commonly measured in pounds of active ingredient/acre. The amounts of herbicide which may be present in air, water, and soil as a result of these operations are usually expressed in parts of herbicide per million parts of medium (air, water, or soil). Although it is possible to accurately measure the amounts of herbicide used in a particular vegetation management program, it usually is not possible to account totally for the ultimate fate and distribution of any particular substance or its degradation products.

The continual need to practice vegetation management is strong evidence that herbicides are neither absolutely effective nor eternally persistent. Phytotoxic amounts of herbicides persist for discrete periods which are determined by their rates of degradation under specific environmental conditions. Some factors which act to reduce herbicide concentrations in particular environmental compartments (air, water, soil, plants) are discussed in this section.

Steady loss of herbicide residues in soil results from microbial and chemical degradation, adsorption, leaching, volatility, and photodecomposition. The principal soil microorganisms are algae, fungi, actinomyces, and bacteria. They must have food for energy and growth. Organic chemicals in soil provide this food supply, except for a few organisms that utilize inorganic substances. Herbicidal chemicals are among the types of degradation matter that can be degraded and/or utilized by soil microorganisms. Favorable environments for soil microorganisms have high organic matter contents, are moist, warm, well-aerated and well-supplied with mineral nutrients (e.g., nitrogen, phosphorous, potassium). Under favorable conditions where soil microorganisms are abundant, organic chemicals including herbicides are short-lived. Microbial degradation is the principal means of reducing soil residues of herbicides.

High rates of herbicide application may result in qualitative and quantitative changes in populations of soil microorganisms. High rates are used for vegetation control at utility substations where "soil sterilization" is desired. Elimination of soil microorganisms, in effect, extends persistence of herbicides in soil and makes it possible to use lower amounts of herbicide over time for complete vegetation control.

Chemical processes may reduce soil levels of herbicides or their degradation products. These processes involve reactions between herbicide molecules and other naturally-occurring chemicals, which alter the structure and therefore the properties of the herbicide molecules. Important chemical reactions which alter molecular structures of herbicides include oxidation, (i.e., combining with oxygen), reduction (i.e., combining with hydrogen), and hydrolysis (i.e., combining with water molecules). Usually these chemical processes are much less important in the reduction of soil residues than microbial activity.

Soils containing high amounts of organic matter and clay have a high capacity to adsorb (i.e., take up and hold) organic herbicides. Small particles which have extremely large surface areas in proportion to their volume are especially important in holding organic substances. Adsorbed (bound) herbicides exist in equilibrium with herbicides in the soil solution. Only unbound herbicides in the soil solution are available for adsorption by plants, microbial and/or chemical degradation, and leaching. Soil adsorption tends to decrease phytotoxicity but increase the persistence of herbicides at a particular site.

Leaching is the downward or lateral (off-target) movement of an herbicide in solution through the soil. Soil adsorption,

water solubility, and the amount of rainfall are the factors which determine the extent of leaching. If water evaporates from soil surfaces, herbicides may migrate upward and be deposited as the water evaporates. Lateral migration of water and herbicide may also occur. Leaching should not be confused with surface runoff. If rainfall occurs soon after application of herbicides, surface runoff may reduce soil residues. Steep slopes, low soil permeability, and lack of ground cover tend to promote surface runoff of herbicides.

Soil residues also may be reduced by volatilization, vapor phase movement in soil, and evaporation at the soil surface. Dichlobenil is an herbicide used by BPA which must be incorporated into soil to avoid losses via volatilization. Photodecomposition results from the absorption of ultraviolet sunlight. Although many light-catalyzed reactions are known, the practical significance of this process is uncertain. Photodecomposition is not an important means of reducing soil residues of herbicides used by BPA.

Water and air are fluids which can be important in the movement of herbicides. Most of the herbicides used by BPA are applied to target vegetation through the air, but the amounts escaping to the environment via that route are uncertain. Controlling the drift of small droplets (aerosols) and controlling the formation of vapor (volatilization) is necessary to minimize harmful effects on unintended targets such as vegetation and bodies of water. Herbicide impacts on water are minimized by strict observance of buffer zones and drift control measures as described in the BPA Transmission Line Maintenance Standard.

Toxicity testing and risk assessment are based upon the biological activity of the parent compound. The parent compound is invariably present in greater concentration than any of the primary or secondary degradation productions. Degradation products are tested only when special concerns about possible health effects are present. Two examples of testing of chemicals related to the parent compound are studies conducted using possible 2,4-D contaminants and others with hypothetical bromacil products (see respective background statements). In neither of these cases did toxicity testing reveal toxicity in excess of that of the parent herbicide.

It is important to recognize that knowledge concerning environmental fate and chemical toxicity is being developed and reported on a continuing basis. Efficient and effective use practices at any given time represent the best practicable methods according to data gathered by evaluating herbicide performance under actual field conditions and by carefully controlled laboratory studies.

Chemical Toxicology in Animals and Humans

Toxicity is defined as the inherent capacity of a substance to produce harm or injury in a living organism. Toxicity response in an organism is described as acute or chronic, depending on the amount and length of exposure. Acute toxicity is a fairly rapid response (i.e., death, skin irritation) of an organism to a few relatively large doses of chemical administered over a short period of time. In contrast, chronic toxicity is a slow or delayed response in an organism, or in an organism's offspring, to exposure over a relatively long period of time (U. S. Forest Service 1981, Norris 1971). A common misconception is that "toxicity" or "poisoning" must refer to death, when in fact these terms do not necessarily mean that death must result. A toxic or poisonous substance is a chemical that impairs, injures, or kills an organism by interfering with a body or cellular function.

The toxicity of a particular herbicide can be established readily and compared with that of other chemicals by appropriate tests using laboratory animals. This testing is a fundamental part of the continuous process of hazard assessment. As noted previously, hazard assessment also must consider the pattern of use of the herbicide and the resulting magnitude and duration of animal and human exposure.

The landmark measurement of toxicity in animals is the acute oral LD50 (lethal dose 50). The LD50 is expressed in units of mg chemical per kilogram body weight. The LD50 is the dosage (mg/kg) required to kill 50 percent of a test population of animals (usually rats). These testing procedures utilize single doses and the oral route of exposure; animals are usually observed for short periods of time, most often 24 hours. LD50 data are available for a very large number of chemicals, and it is the most basic information about the toxicological activity of chemicals.

Aquatic organisms are not fed a chemical, but rather are exposed to a chemical in the water. As a result, the landmark measure of toxicity in aquatic animals is the LC50 (Lethal Concentration 50). The LC50 may be expressed in units of mg chemical per liter of water (mg/l), which is equivalent to unit parts per million (ppm), e.g., 10 mg/l = 10 ppm.

Occasionally one will find in toxicological literature a reference to a test dose fed to birds or mammals, but expressed in ppm. This refers to the ratio by weight of the chemical to the food or water in which it is mixed.

Skin and eye irritation are also evaluated using rabbits early in toxicity testing. Skin sensitization is an allergic response that is usually studied in guinea pigs. Measurements of inhalation toxicity are also made using laboratory animals.

Chronic toxicity testing is performed using two-year feeding studies in rats and dogs. Doses are selected to identify a probable no-effect level as well as at least two higher levels of exposure. The general condition, food consumption, growth, behavior, blood and clinical chemistry of the test animals are regularly monitored. Animals are killed at the end of the study and gross and microscopic examination of tissues is performed. This examination will reveal target organ toxicity and carcinogenic activity (i.e., capability to induce cancer) of the compound being tested.

Three-generation reproduction studies are conducted using rats fed diets containing a test chemical. Data concerning reproductive performance, including fertility, gestation, viability, and lactation, are collected. Pups are first examined grossly, then tissue clearance is performed to establish the integrity of the skeletal system, thereby determining the teratogenic potential (i.e., capability to produce birth defects) of the test chemical.

Mutagenicity testing is another element of toxicity testing programs. Mutagenesis is the alteration (i.e., mutation) of inherited genetic material (i.e., alteration of DNA in the paternal or maternal reproductive cells). Various tests have been used to assess the capability of a chemical to alter inherited genetic material. Cytogenetic tests utilize mammalian cells cultured with a test substance; the chromosomes (genetic material) in the cells are examined for breakage and failure to function normally during the stages of cell division. Host-mediated tests employ microorganisms given to a host (usually a mouse) which is subsequently treated with a test chemical. The microorganisms are subsequently reclaimed and examined for changes in growth. "Dominant lethal" tests utilize treated males that are mated with untreated females. Later, the females are killed and the number of dead or resorbed fetuses is used as an index of mutagenicity. The presence of dead or resorbed fetuses indicates that mutations have occurred in the sperm cells, since the females were not treated.

In vitro microbiological assay systems (i.e., outside a living body and in an artificial environment) used to examine chemicals for mutagenicity are relatively recent additions to toxicity testing programs. Bacterial organisms including Salmonella typhimurium (laboratory strains: TA 1535, TA 1537, TA 1538, TA 98, and TA 100); Escherichia

coli (WP2); repair-deficient and repair-proficient strains of Bacillis subtilis (H 17 and M 45) and of E. coli (W 3110 and p 3478); and the yeast Saccharomyces cerevisiae (D3) are used. These assay systems may be supplemented with tests on rat liver enzymes to provide information about chemical effects of metabolic degradation products. The goal of studies with these organisms is identification of mutagens and carcinogens. Batteries of tests are usually used and results are presently difficult to relate to risk assessment.

Radio-labeled chemicals (usually ^{14}C- or ^{3}H-) are used to measure the uptake, distribution, metabolism, and excretion of test chemicals in experimental animals. Species-to-species differences are usually quantitative rather than qualitative. Knowledge gained from studies of this sort is important in determination of the consequences of accidental, unintentional, and unavoidable human exposures.

A very limited amount of information is available concerning direct effects in humans. In most instances, data are limited to brief case reports resulting from accidental exposures. These few cases have validated the use of animals such as rats, rabbits, and guinea pigs in toxicity testing.

Epidemiological studies of the health impact of herbicides are extremely limited in both number and scope. Epidemiological studies are conducted to determine the subtle, long-term effects of exposure; effects which may not appear until many years after exposure or many years of exposure. Epidemiological studies normally require many hundreds, if not thousands, of people exposed to an herbicide before meaningful conclusions can be made as to whether a real difference exists bwtween the health of those people exposed and of those never having been exposed to the herbicide under consideration. Furthermore, a perceived difference may not be easily attributed to the herbicide in question unless the lifestyles, exposures to other chemicals, etc. are similar for all test subjects. It is quickly obvious that epidemiological studies are difficult to conduct. With concern for maintenance of safe working conditions, persons engaged in various aspects of herbicide use have been studied and continue to be studied. What information is available is discussed in each of the Herbicide Background Statements.

Potential Impact on Nontarget Organisms

This section of the Background Statements includes consideration of vegetation, fish and aquatic organisms, wildlife, and livestock that may be exposed inadvertently to herbicides. Toxicological impacts on fish and aquatic organisms,

wildlife, and livestock are investigated as extensions of acute and chronic toxicity testing. Potential impacts are also considered in view of the potential for bioaccumulation. In the absence of bioaccumulation studies or evidence of bioaccumulation, acute or chronic effects from direct exposure are used to assess potential impacts. Nontarget vegetation adjacent to treated rights-of-way or other BPA facilities is inherently susceptible to the herbicides used in vegetation management. Drift is an ever-present concern in aerial and broadcast application of herbicides. The possibility of drift can be minimized by careful attention to maintenance of equipment and environmental conditions and by use of special drift control additives, as detailed in the BPA Transmission Line Maintenance Standard.

Hazard Assessment for Current Use Practice

The final section of the Background Statements reviews the pattern of use of the particular herbicide by BPA and the associated hazard to humans or the environment. The classification of each of the herbicides is indicated using the Human Hazard Signal Word System (40 Code of Federal Regulations 162.10). The Signal Word classification is of most value to persons engaged in handling and application of the herbicides. The environmental fate and results of toxicity testing are briefly reviewed. When results of epidemiological studies are available, they are included.

Finally, the degree of hazard to personnel, the public and the environment from use of the herbicide in the manner proposed by BPA is indicated. The degree of hazard is generally classified as "no hazard", "low", "moderate", or "high". These general categories are based on assessing the estimated risk to a population of individuals, whether it be the public, BPA personnel, or populations of fish and wildlife species. Risk assessment includes not only the toxicity (acute) of the herbicide, but also includes the probability of exposure, the probable duration of exposure, the probable degree of exposure, and the probability of a catastrophe weighted against the benefits achieved. In a programmatic situation, the actual calculation of probable risk is not possible. Risk assessments for herbicide applications can only be made with great effort on a case-by-case basis.

The information developed in the Herbicide Background Statements is summarized and discussed in the section of Chapter 7 ENVIRONMENTAL CONSEQUENCES on Public Health and Occupational Safety.

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AMMONIUM SULFAMATE

Chemical Identification

Ammate is the common name for ammonium sulfamate. Ammate X and Ammate X-NI are the trade names of the two formulations manufactured by E.I. Du Pont de Nemours and Company (Inc.). The EPA registration number for Ammate X is 352-206-AA and the registration number for Ammate X-NI is 352-311. Ammate X contains sodium dichromate, a rather toxic corrosion inhibitor, and is not cleared for use adjacent to water supplies. Ammate X-NI lacks the corrosion inhibitor and is cleared for use right up to the water's edge.

Ammonium sulfamate is an odorless, crystalline solid. Technical ammate is yellow. Decomposition occurs at 160^{o}C. Ammonium sulfamate has negligible vapor pressure. The crystals absorb water when exposed to air. Ammate is very soluble in water (216 g/100 ml).

Action in Vegetation

Ammate is an inorganic herbicide which is especially effective in control of woody plants (hardwoods, shrubs, and conifers). Annual and perennial herbaceous plants are also sensitive to ammonium sulfamate.

Ammate is rapidly absorbed by plant foliage and stems. Little is known of the mechanism of action of inorganic herbicides, including toxicity. Ammonium sulfamate prolongs the dormant period of plants. During the prolonged dormant stage, starch and sugar reserves are exhausted, which may be associated with death of the plant (Robbins et al. 1952).

Utiization by BPA

Ammate X-NI is used by BPA on rights-of-way and at substations for the management of weeds and brush near surface water. BPA used 969 pounds of ammonium sulfamate (active ingredient) between 1978 and 1981. None was used in 1979 and none is proposed for use in 1983.

Ammate X-NI is diluted with water and applied to foliage with portable sprayers or high pressure hydraulic hose sprayers during the full leaf stage. Dilution provides a

spray concentrate of 395 to 575 grams ammonium sulfamate (active ingredient) per liter. Directed application of ammate crystals or the aqueous solution (395-575 g/l) also can be made to cut stumps or during single-stem treatments.

Chemical Fate and Distribution in the Environment

Soil

An early report (Crafts 1945) indicated that ammonium sulfamate (AMS) is not retained in soil but moves with soil moisture like sodium chlorate. Crafts (1945) also suggested the sulfate and nitrate released by AMS breakdown acted as plant nutrients and stimulated growth at low rates of application.

Strains of two fungi (Aureobasidum pullulans, Aphalosporium acremonium) and two unidentified species of bacteria (Achromobacter sp., Flavobacterium sp.) can utilize sulfamic acid (sodium salt) as a source of nitrogen. The fungi and bacteria converted the sulfamate to sulfate in the same proportion as the amount of nitrogen assimilated (Jensen 1963).

Under humid conditions, AMS applied at a rate of 3 pounds/1,000 square feet inhibited germination of weed seeds, but phytotoxicity in various grasses and crops disappeared after six to eight weeks (DeFrance 1943).

Water

The fate of AMS in water has not been reported. Due to its high water solubility, any AMS in surface runoff would be rapidly diluted. Ammate X-NI is designed specifically for controlling undesirable vegetation growing on land adjacent to and surrounding domestic water supply reservoirs, supply streams, lakes and ponds (Du Pont, no date).

Air

The fate of AMS in air has not been reported. Photodecomposition is not important in the degradation of AMS.

Chemical Toxicology in Animals and Humans

Acute Effects

The acute oral LD50 of ammonium sulfamate in rats is 3,900 mg/kg. It therefore is classed as a slightly toxic chemical

(Du Pont 1972). Vinokurova and Mal'kova (1963) reported that the acute oral toxicity of AMS was 4,400 mg/kg in rats and 3,100 mg/kg in mice.

Because of the important flame retardant properties of AMS, studies of skin irritation and sensitization in animals and humans has been conducted. Neither skin irritation nor systemic toxicity were observed in rats following repeated application of 20 percent or 50 percent aqueous ammonium sulfamate solutions (WSSA 1974; Du Pont 1972). Skin irritation tests were done in rats and rabbits treated daily for 20 days with 15, 20, or 30 percent solutions of AMS. One rat showed erythema (skin inflammation) on day 7 but no irritation during the remainder of the test. No histopathology was evident in rats. Slight atrophy of the epidermis was reported in the rabbits (Du Pont 1981).

Results of testing in humans have been consistent with findings in rats, rabbits, guinea pigs, and mice. Tyvek fiber containing 45 percent AMS produced mild irritation in 5 of 191 human subjects who wore test patches of the material for six days. One person of 205 subjects showed mild irritation after six days to a cotton denim patch containing 15 percent AMS. No evidence of allergic sensitization was seen in these two test groups (396 human subjects) when a second application of the patches (Du Pont 1981) was made 10 days after completion of the first test.

Aqueous solutions instilled into the conjunctival sac of the eyes of five rabbits (0.5 ml of a 4 percent solution) and applied to the mucous membrane of a rabbit's eye (drop of a 20 percent solution) did not produce ill effects (Du Pont 1981).

Inhalation of AMS dusts can produce ill effects. The threshold limit value (TLV) is 10 mg/m^3 and the short-term exposure limit (STEL) for 15-minute excursions is 20 mg/m^3 (ACGIH 1976). The OSHA standard is 15 mg/m^3 (Du Pont 1981). Laboratory workers exposed to 100 to 200 mg/m^3 perceived a tickling or itching in the throat and a tingling in the nose (Du Pont 1981).

In one method of interpreting toxicity data, it is presumed that acute (LD50) toxic doses for rat populations are similar to that for human populations. Given an LD50 of 3900 mg/kg for rat populations, it is presumed that the LD50 dose of ammonium sulfamate (active ingredient) for a population of 165-pound humans would be 10.3 ounces (0.063 oz/lb).

Chronic Effects

Rats fed diets containing 1 percent ammonium sulfamate (10,000 ppm) for 105 days exhibited no clinical or histological signs of toxicity. Diets containing 2 percent ammonium sulfamate inhibited growth but did not induce histopathology (Ambrose 1943).

Du Pont (1981) lists ten additional extended studies of chronic toxicity of AMS under laboratory conditions. Uniformly, a low order of toxicity was observed. Most of the studies are proprietary work conducted by the manufacturer.

Ten rats orally given 1 ml of a 50 percent AMS solution on alternate days were killed after 9 or 15 treatments. Growth rate was initially reduced, but the rats gained weight normally during the latter part of the test period. No other signs of toxicity were observed and neither gross nor microscopic lesions were observed in tissues.

Six male rats were given a daily oral dosage of 2,200 mg/kg five times a week for two weeks. Animals maintained good condition including normal body weight except for occasional diarrhea. Three rats were killed after the tenth treatment and showed evidence of chronic gastritis. When the other rats were killed 10 days later, no evidence of gastritis was present.

No toxicity was observed in a dog fed 1,000 mg AMS daily for 13 days.

AMS was fed for 90 days to groups of 32 rats at dietary levels of 350 and 500 ppm. No clinical signs of toxicity were observed and tissue histopathology was not remarkable.

Cupta el al. (1979) published results of 90-day toxicological studies in the rat. Rats were given either 0, 100, 250, or 500 mg/kg for six days per week. No adverse effects were observed with respect to appearance, behavior, or survival. Weight gain was reduced at the highest dosage after 60 days. Food consumption was gradually reduced and water intake increased in all treated rats. No significant changes in relative organ weights were observed. Hematological studies at 30, 60, and 90 days did not reveal significant changes. The liver of one rat at the highest dose after 90 days showed slight fatty degenerative changes. Throughout the study, the general condition and health of the animals remained apparently good.

No evidence of carcinogenicity was obtained in studies in which rats were fed either 350 or 500 ppm AMS in their diet for 19 months (Du Pont 1981).

Ammonium sulfamate was included in an evaluation of possible mutagenic activity of 100 herbicides. Ammate did not induce point mutations in eight strains of histidine-requiring mutants of the bacteria Salmonella typhimurium (Anderson et al. 1972).

Potential Impact on Nontarget Organisms

Vegetation

Ammonium sulfamate is an effective contact herbicide and spray or drift will injure the fruit or foliage of nontarget species. Such damage will be limited by the degree of exposure, environmental conditions (particularly temperature and the amount of moisture), and the innate sensitivity of the species.

Fish and Other Aquatic Organisms

These facts were taken from a summary provided by Du Pont (1981). Ammonium sulfamate has a low order of toxicity to fish. The 24-hour, 48-hour, and 96-hour LC50s of AMS to fingerling channel catfish were 259, 206, and 203 ppm (Clemens 1959). Young carp were not killed by 48-hour exposures to 500 ppm AMS and the median threshold limit was 1,000 to 2,000 ppm (Maki 1973). Rainbow trout, bluegills, and sea lampreys survived a concentration of 5 ppm for 24 hours in Lake Huron (Applegate el al. 1957).

Ammonium sulfamate (30 ppm) applied to the head of an artificial stream had no effect on the aquatic community composed of bottom-dwelling and drifting invertebrates and rainbow trout (Maki 1973).

Wildlife

A low order of toxicity would be expected due to results of studies of rodents, fish, and livestock. Limited data support this generalization.

The acute oral toxicity of AMS in quail is 3,000 mg/kg (Du Pont 1981). Body weight gain and the general condition of quail were not affected by feeding AMS diets to give 150 and 590 mg/kg/day for 14 days. The fertility of quail was not affected by two 10-day periods of feeding diet containing AMS (dosage 150 mg/kg/day).

AMS-treated sweet gum and post oak (Quercus stellata) were not toxic to deer (Haugen 1953). Additional feeding trials

were conducted using crystalline ammonium sulfamate and three other species of trees.

Livestock

No adverse effects were reported in cattle and sheep fed AMS or diets containing AMS in high amounts (Belasco 1954; Haugen 1953; and other references in Du Pont (1981).

Acute toxicity of AMS is of a low order in livestock. No fatalities of sheep (yearling) occurred following oral administration of 14.1 and 28.3 grams/animal. Similarly, pregnant ewes delivered normal lambs following oral administration of 112 grams AMS/sheep. Oral doses of 100 grams were lethal to goats (Du Pont 1981).

Daily oral administration for ten days of 28.3g AMS did not affect the condition of calves (approximate daily dosage 0.42 grams/kg. No ill effects were observed in sheep given 225 grams AMS (total) in a five-day period (Du Pont 1981).

Hazard Assessment for Current Use Practice

Ammonium sulfamate is used to control brush beneath transmission lines and towers. Ammonium sulfamate is nonvolatile and virtually nontoxic to humans or livestock. As a result, it may be applied to weeds adjacent to crops or to unwanted vegetation near bodies of water.

Based upon testing in laboratory animals, particularly acute oral and inhalation toxicity and skin and eye irritation, the Human Hazard Signal Word CAUTION appears on the herbicide label. Ammonium sulfamate has a very low order of toxicity in animals and humans. Because of its potential use as a fire proofing agent in cloth, ammonium sulfamate has been subjected to more direct testing in humans than have most other herbicides. The results of these tests are consistent with data obtained in work with animals.

No evidence of carcinogenic, mutagenic, or teratogenic activity has been obtained.

Ammonium sulfate may break down in the environment to nitrogen and sulfate which can be utilized as nutrients by microorganisms and plants.

The use of ammonium sulfamate as directed on the pesticide labels results in a low degree of hazard to personnel, the public, and the environment.

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ATRAZINE

Chemical Identification

Atrazine is the common name for 2-chloro-4-ethylamino-6-isopropylamino-s-triazine. AAtrex is the trade name of atrazine herbicide that is marketed by the Agricultural Division of Ciba Geigy Corporation (Greensboro, North Carolina). Two herbicidal formulations of atrazine are used by BPA, AAtrex 80W (EPA Reg. No. 100-439) and AAtrex Nine-O (EPA Reg. No. 100-585).

Atrazine is a white, crystalline solid that melts at 173°C to 175°C. The vapor pressure of atrazine is 3.0×10^{-7} mm Hg at 20°C, and 2.2×10^{-5} mm Hg at 50°C. The solubilities of atrazine (parts per million by weight) in dimethyl sulfoxide, chloroform, ethylacetate, and methanol are 183,000, 52,000, 28,000, and 18,000, respectively. The limit of water solubility of atrazine is 33 ppm. Atrazine is subject to decomposition by ultraviolet light (WSSA 1979).

Action in Vegetation

Atrazine is registered for use in the control of annual and perennial grasses and broadleaf weeds in corn, sorghum, rangeland, macadamia orchards, pineapple, and turf grass sod. It is also used for selective weed control in conifer reforestation, Christmas tree plantations, and grass weed fields, as well as for nonselective control of vegetation in chemical fallow (WSSA 1979).

Absorption of atrazine occurs through both the roots and aerial parts of plants. Foliar absorption of atrazine is usually not important under field conditions. The herbicide can be washed off treated plant foliage by rain (WSSA 1979).

The mode of action of atrazine is discussed in detail by Ashton and Crafts (1973). Atrazine inhibits the growth of all organs of intact plants. This effect results from a deficiency of photosynthate due to atrazine's potent inhibition of photosynthesis.

The fate of atrazine in plants is also reviewed by Ashton and Crafts (1973). Hydroxylation and dealkylation of atrazine are important metabolic processes. Shimabukuro and Swanson (1969) reviewed atrazine degradation with respect to the selective toxic action of atrazine.

Utilization by BPA

Atrazine is used by BPA in noncrop areas at substations for the control of weeds along fence lines. BPA used 132 pounds of atrazine (active ingredient) between 1979 and 1981. None is proposed for use in 1983.

AAtrex 80W and AAtrex Nine-O are mixed with water and applied with portable sprayers to weeds just before or soon after growth begins. Dilution and application rates provide an effective application rate of atrazine (active ingredient) between 536 and 4,480 mg/m^2, depending on the susceptibility of the target vegetation.

Chemical Fate and Distribution in the Environment

Soil

"Atrazine is more readily adsorbed on muck or clay soils than on soils of low clay and organic matter content. The downward movement or leaching is limited by its adsorption to certain soil constituents. Adsorption is not irreversible and desorption often occurs readily, depending upon such factors as temperature, moisture, and pH. Atrazine normally is not found below the upper foot of soil in detectable quantities, even after years of continuous use." (WSSA 1979).

When atrazine, simazine, or propazine was surface applied and leached, most of the herbicide remained in the first inch of soil regardless of whether 2, 4, or 8 inches of water were applied. Atrazine is the most water soluble of the three s-triazines and leached to the greatest extent to a six-inch depth (Kozlowski and Kuntz 1963).

Kearney et al. (1969) considered the persistence of pesticide classes, including the triazines. They ranked chlorinated hydrocarbon insecticides as most persistent, and organophosphate insecticides as least persistent. Triazines were generally of intermediate persistence with 2 to 18 months being required for degradation.

Chemical hydrolysis of atrazine and other s-triazines has been reviewed (Helling et al. 1971). Since nonphytotoxic 2-hydroxy-s-triazines are formed, the transformation is considered a degradation. This product may constitute 50 percent of the extractable material within two months (Brown 1978).

Microbial metabolism is another route of atrazine degradation in soil. Soil microorganisms, particularly soil fungi, remove N-ethyl groups of atrazine. Ring cleavage of atrazine and s-triazines is a slow process (Kaufman and Kearney 1970).

Water

Atrazine has been measured in surface waters in Iowa where it is extensively used in corn production. Several examples are cited in a review (Brown 1978). Concentrations in drinking water in Des Moines were 2 ppb in June and 0.2 ppb in August.

Runoff water contained 3 ppm atrazine and suspended solids contained 4.5 ppm following the first rain on an Iowa cornfield treated with 3 pounds/acre. This silt loam field had a 10 to 15 percent slope. A sandy loam field with 6 percent slope lost 2 percent of the applied atrazine with a 0.5-inch rainfall. A 2.4-inch rainfall removed 7 percent of the atrazine.

Triplett et al. (1978) studied the movement of atrazine from conventional and no-tillage corn watersheds. Highest concentrations of atrazine (0.48 ppm) were measured in runoff which occurred shortly after application. Later runoff contained lower amounts. A maximum of 6 percent of the applied herbicide was transported from the field. The average for all watersheds was 2 percent. Less runoff and atrazine transport occurred from no-tillage plots than from conventional plots.

Atrazine was found in the New Orleans water supply at concentrations of 4.7, 5.0, and 5.1 ppb. Diethylatrazine, a degradation product, was present at 0.27 to 0.51 ppb (NRC 1977). A survey conducted by the National Research Council (1977) showed that all Iowa water sampled contained atrazine.

Air

"The significance of photodecomposition and/or volatilization of atrazine from soil is not fully understood. Available data indicate that both occur to some extent if high temperatures and prolonged sunlight follow application before precipitation, but that these factors are of little significance in Atrazine dissipation under most field conditions" (WSSA 1979).

Chemical Toxicology in Animals and Humans

Acute Effects

Atrazine has a low order of toxicity in rats. The acute oral LD50 of technical atrazine is 3,080 mg/kg. The acute oral LD50 in mice is 1,750 mg/kg.

The toxicity of a formulated atrazine (80 percent wettable powder) via the dermal route is also very low. The LD50 in rats is 9300 $\pm$ 990 mg/kg.

Rats were exposed to Atrazine aerosols containing 1.8 to 4.9 mg/liter. No deaths or signs of toxicity were observed in rats exposed for one hour. There should be no acute inhalation hazard involved in the casual handling of this formulation (WSSA 1979).

There have been no reports of substantial skin irritation resulting from experimental or commercial applications of atrazine (WSSA 1979).

The above acute toxicity data are part of proprietary information presented by the manufacturer in support of the herbicide registration of atrazine.

In one method of interpreting toxicity data, it is presumed that acute (LD50) toxic doses for rat populations are similar to that for human populations. Given an LD50 of 3080 mg/kg for rat populations, it is presumed that the LD50 dose of atrazine (active ingredient) for a population of 165-pound humans would be 8.1 ounces (0.049 oz/lb).

Chronic Effects

"Two-year feeding studies, in which male and female rats were given daily dosages of atrazine at various levels, showed no gross or microscopic signs of toxicity due to ingestion of levels as high as 100 ppm in the total diet" (WSSA 1979).

Newton and Dost (1981) note that studies of carcinogenicity contracted by the manufacturer and conducted at Industrial Biotest Laboratories were partially lost as a result of the failure of that laboratory. Testing is being repeated and results will be submitted to the EPA as proprietary information.

No evidence of carcinogenicity was detected in two strains of mice given 83 ppm atrazine in their diets for their lifetimes (Innes et al. 1969).

Atrazine has weak potential for causing chromosomal damage based upon dominant lethal testing using fruitfly (Drosophila melanogaster) (Murnik and Nash 1977). Other investigators have reported that atrazine does not cause point mutations (Anderson et al. 1972; Siebert and Lemperle 1974; Plewa and Gentile 1976; Seiler 1973).

Plewa and Gentile (1976) have reported a dose-dependent increase in the mutagenicity of corn plant extracts. The plants were grown in the laboratory in vermiculite and water without nutrients. Bakshi et al. (1981) found that untreated and atrazine-treated corn plant extracts produced mutation in vitro. Younger plants grown in a growth chamber did not contain mutagenic activity. Atrazine may have stimulated growth in the vermiculate-water cultured plants used by Plewa and Gentile.

Barley anthers were soaked for 12 hours in 1,000 ppm atrazine (Wuu and Grant 1966, 1967). Atrazine had a relative mutagenicity of 10 compared to control (1) and 5,500 R x-rays (32) in a barley test system. No follow-up studies by Wuu and Grant or others have been published.

Large-scale screening tests for teratogenicity were conducted by Bionetics Research Laboratories of Litton Industries under a contract from the National Cancer Institute (Mrak 1969). Atrazine was administered subcutaneously in dimethyl sulfoxide to rats at a dosage of 46.4 mg/kg on days 6 through 14 of pregnancy. No significant increase in fetal anomalies was observed.

The fate of atrazine in animals has been extensively studied using several animal models (Esser et al. 1975). Absorption is rapid from the gastrointestinal tract and most of the metabolites are eliminated in urine. Atrazine is not stored in tissues.

Bakke et al (1972a) studied the metabolism of atrazine and 2-hydroxyatrazine in the rat. Seventy-two hours after dosing, urine and feces contained 65.5 percent and 20.3 percent of ^{14}C-atrazine and 78 percent and 5.5 percent of ^{14}C-hydroxyatrazine, respectively. Less than 0.1 percent of either substance was collected in expired air, indicating no cleavage of the ring structure. Atrazine (and metabolites) in tissues constituted 15.5 percent of the dose and 2-hydroxyatrazine (and metabolites) in tissues was 0.1 percent of the dose. Nineteen urinary metabolites were separated in the atrazine studies including 2-hydroxyatrazine and its mono-N-deethylated derivatives.

Corn and sorghum metabolize atrazine to methanol-insoluble residues (up to 52 percent of the radioactivity present after 336 hours) which were fed to sheep and rats. Both species eliminated these residues in feces (sheep, 100 percent; rats, 88 to 93 percent) indicating poor absorption of these unextractable atrazine derivatives (Bakke et al. 1972b).

These studies illustrate the pathways of biotransformation and rapid elimination of atrazine.

Potential Impact on Nontarget Organisms

Vegetation

Atrazine is an important selective herbicide in vegetation management. It is used to control annual grasses and broadleaf weeds in corn and several other crops. It may be applied as a preemergence herbicide to annual weeds. This pattern of use indicates that atrazine can be used selectively.

Selective weed control in conifer reforestation and Christmas tree plantations requires lower application rates than nonselective weed control on noncroplands.

Tolerance to atrazine may result from reduced absorption and translocation or, more commonly, enhanced degradation. Reduced absorption and translocation have been observed in atrazine-tolerant fall panicum and green foxtail in cornfields (Kern et al. 1975). Ashton and Crafts (1973) reviewed examples of tolerance resulting from rapid atrazine degradation in higher plants, the classical example being corn.

Resistance of weeds to atrazine and simazine has been documented in Washington state. Common groundsel treated since 1958 was tolerant to s-triazines in 1968 (Ryan 1970).

Fish and Other Aquatic Organisms

Fish and aquatic invertebrates are sensitive to prolonged exposures to atrazine in parts per million concentrations. Such levels are extremely unlikely to result from use of atrazine herbicide.

The 48-hour LC50 of atrazine in rainbow trout was 12.6 ppm and the 24-hour LC50 in harlequin fish was 0.55 ppm. Spot exposed to 1 ppm for 48 hours exhibited no deleterious

effects. Bluegill and green sunfish fry survived a three-day test in 10 ppm atrazine (Pimentel 1971).

Bathe et al. (1974); cited in Newton and Dost, 1981) tabulated 96-hour LC50s in various fish species. Values ranged from 5 to more than 100 ppm.

In pond enclosures, concentrations of 0.5 to 2 ppm reduced clam populations and increased the numbers of snails (Walker 1962).

Shell growth of eastern oysters was not affected by 96-hour atrazine exposures of 1 ppm (Butler 1963).

The 48-hour LC50 of atrazine in water fleas (Daphnia magna) was 3.6 ppm (Pimentel 1971). He further listed results of pond studies (Walker 1962) in which at least 50 percent decreases in the numbers of waterbugs, mayfly nymphs, horse-fly larvae, common midges, mosquitoes, phantom midges, biting midges, caddisfly larvae, oligochaetes, and leaches were observed following exposure to atrazine at 0.5 to 2.0 ppm.

Using a three-stage microcosm consisting of algae, a small crustacean, and fish, Ellgenhausen et al. (1980) studied the uptake (bioaccumulation) of atrazine and other pesticides. Atrazine was rapidly taken up by algae and concentrated 80 times above the level in water (0.5 ppm). Algae desorbed atrazine very rapidly (apparent half-time of five minutes). The crustacean (Daphnia) accumulated ambient concentrations but eliminated the herbicide less readily (five hours versus five minutes). At ambient concentrations of 0.01 ppm, catfish accumulated 0.05 to 0.06 ppm and had an elimination half-time of about 4.5 hours. Gunkel and Streit (1980) have also reported rapid uptake and elimination of atrazine by fish.

These studies indicate that atrazine has little tendency to be bioaccumulated in animals. Thus, when the health of aquatic organisms is at issue, effects as a consequence of direct exposure rather than effects resulting from bio-accumulation must be considered.

Wildlife

Using two-week-old mallard ducklings, pheasants, and bobwhite quail, dietary LC50s of greater than 5,000, and 700 to 800 ppm, respectively, were estimated. Birds were fed for five days and observed for three days in these tests listed by Pimentel (1971).

The oral LD50 of atrazine in mallards was greater than 2,000 mg/kg in another study (Tucker and Crabtree 1970).

Newton and Norris (1968) measured atrazine in edible tissues of deer from atrazine-treated forest lands. The maximum found in any tissue that would be used as food was 0.076 ppm. The rapid metabolism of atrazine and/or the failure of deer to absorb atrazine residues from treated plants may be the basis of this finding.

Livestock

The toxicity of atrazine to cattle, sheep, and chickens has been studied by Palmer and Radeleff (1969). The toxic dosage for cattle was 25 mg/kg after eight doses by drench (water-diluted formulation administered orally) and after two doses by capsule. The toxic dosage for sheep was 5 mg/kg. Ten dosages of 50 mg/kg produced significant reduction in weight gain in chickens. Signs of poisoning in cattle and sheep were muscular spasms of varying intensity in the hind quarters, stilted gait and stance, and anorexia. At necropsy, petechiae (minute hemmorrhagic spots) on the surface of the epicardium and congestion of kidneys, liver, and lungs were generally present.

Application rates of less than 1, 3, and 6.4 pounds/acre would render feed toxic to sheep, cattle, and chickens, respectively (Palmer and Radeleff (1969).

Hazard Assessment for Current Use Practices

Atrazine is a widely used herbicide that is deployed by BPA to a very limited extent for the control of grasses and broadleaf weeds at substations. Broadcast or directed sprays are used which result in small amounts of release of herbicide into the general environment. The potential of nontarget exposures is virtually nil based upon this pattern and extent of use.

Extensive laboratory and field studies have shown that atrazine is moderately persistent in soils, and at higher rates of application may produce soil residues which retain phytotoxicity for a year or more. Atrazine is adsorbed by soil organic matter. Microbial and chemical degradation are primarily responsible for the loss of atrazine from soil. Since atrazine is tightly soil-bound by organic matter and has low water solubility, leaching removes small amounts slowly from treated areas.

Based upon testing in laboratory animals, atrazine has a low order of toxicity. As a result, CAUTION is the Human Hazard Signal Word displayed on the herbicide label.

Atrazine is rapidly metabolized to a large number of metabolites which are readily excreted by animals. As a result, chronic toxicity of atrazine is low. Atrazine has weak potential for causing mutagenic effects based upon dominant lethal tests in fruitflies (Murnik and Nash 1977) but it did not produce point mutations in several test systems (Newton and Dost 1981). No reports of teratogenic or carcinogenic activity have appeared in the scientific literature.

In over 30 years of use, atrazine has no history of causing worker injury.

Use of atrazine as indicated on the pesticide label results in a low degree of hazard to personnel, the public, and the environment.

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BROMACIL

Chemical Identification

Bromacil is the common name for 5-bromo-3-sec-butyl-6-methyluracil. Three herbicides used by BPA contain this active ingredient; Hyvar X-L (E. I. Du Pont de Nemours & Co.), Krovar I (E. I. Du Pont de Nemours & Co.), and Oxy Ureabor (Occidental Chemical Company) contain 21.9, 40, and 1.5 percent bromacil, respectively.

Hyvar X-L Weed Killer (EPA Reg. No. 352-346-ZA) is a water soluble liquid which contains two pounds of the lithium salt of bromacil per gallon. Hyvar X-L is a combustible mixture which should be kept away from heat and open flame. Bromacil is the only active ingredient in Hyvar X-L.

Krovar I Weed Killer (EPA Reg. No. 352-352-AA) is a light brown powder with negligible vapor pressure and dispersible in water. A separate Background Statement on diuron, the other active ingredient in this product, has been prepared (page A-67).

Oxy Ureabor (EPA Reg. No. 10659-51) is an off-white, odorless, granular material that is nonvolatile and water soluble. A separate Background Statement on borate and chlorate, the other active ingredients in this product, has been prepared (page A-131).

Bromacil is a white, crystalline solid. It is odorless and melts at 158°C to 159°C. Sublimation occurs just below the melting point. The vapor pressure of bromacil is 2.5×10^{-7} mm Hg at 25°C and 0.8×10^{-3} mm Hg at 100°C. Bromacil is stable in water, aqueous base, and common organic solvents. Rapid decomposition occurs in acid. Bromacil is soluble in acetone (20.1 g/100 g), absolute ethanol (15.5 g/100 g), acetonitrile (7.7 g/100 g), and xylene (3.3 g/100 g). The limit of water solubility is 815 ppm at 25°C.

Action in Vegetation

Bromacil is used on noncropland areas for control of a wide range of annual and perennial grasses and broadleaf weeds, and certain woody species. Many annual grasses and broadleaf weeds are controlled at rates of three to five pounds/acre. Some perennial weeds and brush species are controlled

at rates of six to ten pounds/acre. Johnson-grass and resistant perennial weeds may require 12 to 24 pounds/acre.

Bromacil is readily absorbed by roots and rapidly translocated to aerial plant parts via xylem. Uptake following foliar or stem application is slower but the process can be enhanced by including a surfactant in the herbicide spray. Root uptake of bromacil in citrus (a crop in which bromacil is used selectively) has been studied by Gardiner et al. (1969).

The primary toxic action of bromacil in plants is inhibition of photosynthesis. Signs of toxicity from bromacil are primarily observable in plant leaves. Depending upon the degree of exposure and species sensitivity, the leaves will appear slightly yellow (chlorotic), which develops slowly, or they will have a water-soaked appearance and become necrotic within a few days (Klingman and Ashton 1975).

Utilization by BPA

Bromacil is used by BPA in noncrop areas at substations and around poles to control weeds and brush. BPA used on the average 666 pounds of bromacil (active ingredient) per year since 1978, and proposes to use 190 pounds of bromacil in 1983.

Hyvar X-L is applied undiluted in spring or summer to root collars with an exact-delivery handgun applicator; effective application rate of bromacil using this technique is about 2,600 mg/stem.

Krovar I is used at substations only, and is applied to the soil surface in spring or summer. Portable sprayers direct an aqueous spray mixture at a rate providing an effective application rate of bromacil (active ingredient) between 179 and 1,792 mg/m^2.

Oxy Ureabor is also used at substations only, but may be applied as the dry formulation with portable spreaders or as an aqueous mixture with portable sprayers. Application to the soil surface is made during the growing season at a rate providing an effective application rate of bromacil between 370 and 2,240 mg/m^2.

Chemical Fate and Distribution in the Environment

Soil

Steady loss of bromacil residues in soil results from volatility, leaching and mobility, photodecomposition, and biological and/or chemical breakdown.

Volatilization is a minor route for the removal of bromacil from treated soils. The volatilization of bromacil from treated soil was less than 0.1 percent per week at elevated temperatures (49°C) (Hill 1971). When radiolabelled ($2-^{14}C$), bromacil was applied to soils in enclosed systems, only $^{14}CO_2$ was volatilized and trapped in a nine-week study. This result indicated extensive microbial breakdown and no losses via volatilization of bromacil from the treated soils (Gardiner et al. 1969).

The degree of soil adsorption of bromacil will determine the extent of leaching and mobility of the herbicide. The K-value is the ratio of the amount of herbicide adsorbed on soil (ppm) to an equilibrium concentration of 1 ppm in water. The higher the K-value, the more strongly adsorbed to soil is the herbicide. The K-value for bromacil is 1.5. Atrazine and diuron are other herbicides used by BPA and their K-values are 2.8 and 5.0, respectively. Thus, bromacil is less tightly adsorbed than either of the other two herbicides. Leaching of bromacil will occur following high rates of application (Skroch et al. 1971). Gardiner (1975) stated that bromacil should be considered neither excessively mobile nor immobile in soil.

Sunlight plays only a minor, or perhaps even insignificant, role in the breakdown of bromacil residues in soil (Gardiner 1975). Bingeman et al. (1962; cited in WSSA 1979) concluded that volatilization and photodecomposition of bromacil from soil were negligible.

Biological and chemical processes are most important in the degradation of bromacil in soil. (Gardiner et al. 1969) measured ^{14}C-bromacil degradation and found that five to six months were required to reduce soil levels to 50 percent of initial concentrations. Degradation to $^{14}CO_2$ as noted earlier, suggests extensive breakdown by soil microorganisms. Of the residual radioactivity in soil, about 90 percent was recoverable in ethanol extracts as intact ^{14}C-bromacil. Minor amounts of hydroxylated metabolites were formed (Gardiner 1975).

Microbiological organic of bromacil and other uracil herbicides has been reviewed (Gardiner 1975). Torgeson and Mee

(1967) reported that a soil isolate of Penecillium paraherquei Abe., a fungus, was particularly active in bromacil degradation. No herbicidal activity was observed in buckwheat 28 days after application of bromacil (12 pounds/acre) to a sterile soil inoculated with P. paraherquei. Zimdahl et al. (1970) have provided evidence of chemical breakdown of bromacil in soil.

Results of studies in Florida (Tucker and Phillips 1970) and Oregon (Migchelbrink 1971) indicate the strong influence of environmental factors, including bromacil organic in reducing soil residues. After five successive applications of bromacil at a rate of 20 pounds/acre/year, only one pound/acre was present 13 months after the last application in the upper 18 inches of a fine sandy Florida soil. In Oregon agricultural soils, a period of two years may be necessary to permit growth of sensitive species such as beans following soil treatments of 0.8 pound/acre to eliminate mint (Migchelbrink 1971).

Water

The possible contamination of surface water following the use of bromacil for nonselective weed control at an industrial site was studied by Davis and Rahn (1970). Surface runoff resulting from heavy rains two to four weeks after application contained insignificant amounts of bromacil. Those amounts were below levels known to be injurious to plants or animals.

In studies of photodecomposition using dilute aqueous solutions (1 to 10 ppm), bromacil and small amounts of 5-bromo-6-methyluracil were recovered following prolonged exposure to sunlight (Moilanen and Crosby 1974).

Air

Volatilization of bromacil from treated soils is not an important process in the reduction of soil residue; therefore, little bromacil should enter the atmosphere.

Chemical Toxicology in Animals and Humans

Acute

Bromacil has a low order of acute oral toxicity in the rat. The LD50 for bromacil (as an active ingredient in an 80 percent wettable powder) in male rats was 5,200 mg/kg. Inhalation exposures of 2.1 and 4.8 mg/liter for four hours did not produce mortality (Zapp 1965).

Administration of large oral dosages of bromacil (5,000 mg/kg) to dogs likewise did not produce mortality in dogs. Excessive vomiting, salivation, weakness, loss of coordination, excitability, diarrhea, and mydriasis (prolonged, excessive pupil dilation) were observed (WSSA 1979).

A 70 percent water paste of bromacil applied to intact, shaved rabbit skin did not cause toxicity or gross tissue pathology. Skin irritation and allergic sensitization studies were conducted in guinea pigs. A 50 percent suspension of bromacil caused mild irritation. No skin sensitization was induced during a three-week test period (Zapp 1965).

Eye irritation studies have been conducted in rabbits. Ten milligrams of bromacil produced mild, temporary conjunctivitis in rabbits. No corneal damage was observed (Zapp 1965).

In one method of interpreting toxicity data, it is presumed that acute (LD50) toxic doses for rat populations are similar to that for human populations. Given an LD50 of 5200 mg/kg for rat populations, it is presumed that the LD50 dose of bromacil (active ingredient) for a population of 165-pound humans would be 13.8 ounces (0.083 oz/lb).

Chronic Effects

Two-week toxicity tests in rats given dosages of 650, 1,035, or 1,500 mg/kg/day, five times/week, resulted in no deaths at the lowest dosage. Five of six survived the intermediate dosage. Four of six rats died at the highest dosage. Livers of rats at each dosage showed hypertrophy (excess growth) and hyperplasia (excess cell proliferation). These effects in survivors were reversible and disappeared after 14 days.

Two-year feeding studies were conducted in male and female rats using dietary levels of 0, 50, 250, and 1,250 ppm. The dietary levels were fixed during the test period. Therefore, the daily dosage decreased as the rats gained weight, e.g., at 1,250 ppm, the bromacil consumption was initially 154.2 mg/kg and finally 38.7 mg/kg. Hematology, urinalysis, and clinical chemistry were conducted at intervals, and were unremarkable. Weight gain in female rats was reduced at the highest dose. Gross pathological lesions were absent. Focal light cell hyperplasia and focal follicular cell hyperplasia were observed in the thyroids of control and high-dose rats. The effect was more prevalent at the highest dose. One follicular cell adenoma (benign glandular

tumor) occurred in a female rat at the highest dose. There were no excessive tissue residues of bromacil.

The above studies, as reported by Sherman and Kaplan (1975), are summarized (EPA 1975) as part of the Substitute Chemical Program.

A 90-day chronic feeding study was conducted by Zapp (1965). Male and female rats (10 each) were fed 0, 50, or 500 ppm for 13 weeks, or 2,500 ppm (weeks 1 through 6) adjusted to 5,000 ppm (weeks 7 through 13) or adjusted to 5,000 ppm (weeks 7 through 10), and to 6,000 ppm (11th week) and 7,500 ppm (weeks 12 and 13). Neither deaths nor signs of toxicity were observed. Reduced weight gain was induced in the two subgroups which began at a dose of 2,500 ppm. Hematology and urinalysis were normal. No pathological lesions were observed at 50 and 500 ppm bromacil. At 5,000 ppm, microscopic changes suggestive of increased thyroid activity were reported (Zapp 1965 as cited in EPA 1975).

Dogs were fed for two years with diets containing 0, 50, 250, and 1,250 ppm bromacil. At the highest dose there was early weight loss, but weights stabilized 1 to 1.5 kg below other dogs in the study. No signs of toxicity were observed. Hematology, urinalysis, and clinical chemistry were not remarkable. Organ weights were not affected by the treatments and no significant tissue accumulation of bromacil was observed (EPA 1975).

The metabolic fate of bromacil has been studied in rats fed for one month with diets containing 1,250 ppm bromacil (Gardiner et al. 1969). Six bromacil metabolites were found in the urine. The primary metabolite was the water soluble conjugate of 5-bromo-3-sec-butyl-6-hydroxy-methyluracil. Minor metabolites included three other hydroxylated derivates, 3-sec-butyl-6-methyluracil and an unknown bromine-containing chemical. 5-Bromouracil (a potent mutagen) is not a bromacil metabolite (Gardiner 1975).

Reproduction studies were included in the Bromacil Report (EPA Pesticide Petition No. 6F0499, Section C) summarized in the EPA review (EPA 1975). Animals which were fed 250 ppm bromacil were used in the three-generation studies. The reproductive performances (fertility, gestation, viability, and lactation studies) of control and bromacil-fed rats were not different. No young were deformed.

In addition to the above study, Paynter (1966) reported results of teratogenicity studies in rabbits fed 0, 50, and 250 ppm from the 8th to the 16th day of pregnancy. On the 28th or 29th day after breeding, three does from the control and 50 ppm groups were killed. Four does on the 250 ppm

diet were killed. After parturition, the remaining does were killed. One-third of the young from each group were cleared and studied for skeletal anomalies. No terata (serious defects) were observed (EPA 1975).

Because of the possibility of formation of 5-bromouracil (a potent mutagen) as a bromacil metabolite, experiments on the metabolic fate and mutagenicity of bromacil have been performed. 5-bromouracil is not found in rat urine or feces (Gardiner et al. 1969) and is not present in urine of bromacil production plant workers (Anonymous 1966). Short-term testing for mutagenicity using a variety of testing protocols have shown that bromacil does not appear to be mutagenic. The absence of mutagenic effects of bromacil has been reported by McGahen and Hoffman (1963, 1966), Anderson et al. (1972), Siebert and Lemperle (1974), Epstein et al. (1972), Ficsor and Nii Lo Piccolo (1972) (references from EPA 1975).

No evidence of carcinogenic activity of bromacil was found in two-year chronic studies in rats or dogs (Sherman and Kaplan 1975; Zapp 1965). An ovarian follicular cell adenoma in a female rat fed 1,250 ppm bromacil was reported by Lawless (1966).

Potential Impact on Nontarget Organisms

Vegetation

Bromacil is used to selectively control weeds by citrus and pineapple producers. Residue tolerances of 0.1 ppm have been established. Bromacil is primarily a broad spectrum, nonselective herbicide and is used primarily in noncroplands. Therefore, residue tolerances do not exist for crops other than citrus and pineapple.

Field applications of bromacil and picloram were less effective than bromacil alone in controlling several weeds including broomsedge, dallisgrass, and several panicums. Laboratory testing using oats showed that picloram reduced bromacil absorption (Sterrett et al. 1972).

The senescence of corn leaves was markedly retarded by sublethal soil residues (0.03 to 0.09 ppm) of bromacil (Hiranpradit and Foy 1973). Plants treated with bromacil exhibited increased chlorophyll retention.

Fish and Aquatic Organisms

Bromacil is slightly toxic to fish. Forty-eight-hours LC50s in bluegills, rainbow trout, and carp are 71, 75, and 164

ppm. Some evidence of cumulative toxicity was obtained in rainbow trout in which the 24-, 48-, and 72-hour LC50s were 1202, 75, and 38 ppm (EPA 1975).

Yoshida and Nishiuchi (1972) reported 48-hour median threshold limits (TLm) of 10 to 40 ppm in carp, Japanese goldfish, and killifish. Other 48-hour TLms were greater than 40 ppm for loach and 230 ppm for tadpoles. The 72-hour TLm for crayfish was 40 ppm and the three-hour TLm for water fleas was greater than 40 ppm.

Hoffman (1972 cited in Ashton and Crafts 1973) reported that monuron-resistant Euglena, a protozoan (single-celled animal), showed similar resistance to bromacil. The resistant strain was not affected at 40 ppm bromacil, and photosynthesis was only partially inhibited at 100 ppm. A wild strain of Euglena was markedly inhibited at 2 ppm and completely inhibited at 10 ppm. Neither strain was sensitive to bromacil in the dark, indicating that inhibition of photosynthesis is one of the phytotoxic actions of bromacil.

Wildlife

The toxicity of bromacil to wildlife is similar to its low order of toxicity to laboratory test species. The eight-day dietary LC50s for mallard ducklings and bobwhite quail were each greater than 10,000 ppm.

Livestock

The toxicity of bromacil to livestock is similar to its low order of toxicity to laboratory test species. Palmer and Radeleff (1969) recorded the toxicity of bromacil and other organic herbicides to cattle, sheep, and chickens. No ill effects were apparent in cattle given capsules or drenched with 10 doses of 100 mg/kg. A single dose of 250 mg/kg produced toxicity in a yearling calf which survived, but sustained a 14 percent weight loss.

Sheep were more sensitive to bromacil (Palmer and Radeleff 1969). Toxicity was observed in sheep given capsules containing 50 mg/kg each day for 10 days. An 8 percent weight loss was reported. Ten doses of a bromacil drench at 100 mg/kg produced toxicity and 9 percent weight loss.

Other studies in sheep (Palmer 1964) revealed that administration of five doses of 250 mg/kg produced tympany (swelling from abdominal gas) and stilted gait within four hours after administration of the first dose. After the last dose, the animals recovered slowly and had marked

lameness. A lower dosage (100 mg/kg) did not produce toxicity after administration of 11 doses but there was an 11 percent weight loss. Palmer and Radeleff (1969) concluded that rates of bromacil application in excess of five pounds/acre would be hazardous to sheep.

Chickens tolerated dosages of 500 mg/kg, with reduced weight gain being the only effect observed. Weight gain was also reduced in chickens given 250 mg/kg, but that parameter was not affected at 100 mg/kg (Palmer and Radeleff 1969).

Tympanites in cattle and sheep given bromacil may result from the effect of bromacil on rumen microflora. High bromacil concentrations (500, 750, and 1,000 ppm) in rumen fluid *in vitro* reduced ciliated protozoans and *in vitro* forage digestibility (Kutcher et al. 1970). Pesticide residues on contaminated feedstuffs would be expected to contain much lower amount of bromacil than those used in the studies of Kutcher et al. (1970).

Hazard Assessment for Current Use Practices

Bromacil is effective for the control of herbaceous and woody plants in BPA vegetation management programs. Bromacil is also an active constituent of Oxy Ureabor, used in soil sterilization programs. In combination with sodium chlorate and metaborate, bromacil improves the effectiveness of Oxy Ureabor against certain perennial grasses.

The Human Hazard Signal Word system is used to classify herbicide products based upon oral, dermal, and inhalation acute toxicity as well as the potential for causing skin and eye damage. Oxy Ureabor is labelled DANGER based upon corrosiveness due to the inorganic active ingredients. Hyvar X-L is labelled WARNING and Krovar I is labelled CAUTION. These bromacil-containing herbicides illustrate the flexibility of the Human Hazard Warning System which is based upon the toxicity of the formulated products rather than the toxicity of individual active formulated ingredients *per se*.

Bromacil is rapidly adsorbed, metabolized, and excreted in animals and does not accumulate in animal tissues. No evidence of carcinogenic, teratogenic, or mutagenic activity has been reported. Specific studies of bromacil metabolism have conclusively demonstrated that 5-bromour-acil (a potent mutagen) is not a product of bromacil degradation in either animals or the environment.

Phytotoxic residues of bromacil persist in soils for a year or more at the high rates of application used in soil

sterilization. Losses of soil phytotoxicity are primarily the result of microbial and chemical degradation rather than the consequence of volatility, photodecomposition, or soil leaching. No significant contamination of water will result from leaching or surface runoff of bromacil.

If nontarget vegetation is inadvertently exposed to bromacil during weed control operations, the degree of exposure will determine the extent of damage. Aquatic organisms, wildlife, and livestock are unlikely to be exposed. In cases of accidental exposure, no harmful effects would be expected due to the low toxicity of bromacil.

Use of bromacil herbicides as indicated on the label and in accordance with the Transmission Line Maintenance Standards results in a low degree of hazard to personnel, the public and the environment.

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DICAMBA

Chemical Identification

Dicamba is the common name for 3,6-dichloro-O-anisic acid (2-methyl-2,6-dichlorobenzoic acid). Five herbicidal formulations containing dicamba are used by BPA. Their trade names and EPA registration numbers are Banvel 4-0.S. (Reg. No. 876-155-AA), Banvel 4-W.S. (Reg. No. 876-159-AA), Banvel 5G (Reg. No. 876-103-AA), Banvel 520 (Reg. No. 876-168-AA), and Banvel 720 (Reg. No. 876-177-AA). Each of these formulations is a product of Velsicol Chemical Corporation. Banvel 4-W.S. and Banvel 720 contain dicamba as a dimethylamine salt; the other three formulations contain dicamba in the acid form.

Dicamba is a white, crystalline solid which is odorless in its pure form. Technical dicamba (i.e., dicamba in the form manufactured for sale) is a brown, crystalline solid. The melting point of dicamba is 114-116°C. Dicamba is resistant to strong alkali and acid and is stable to oxidation and hydrolysis. The vapor pressure of dicamba is 3.75×10^{-3} mm Hg at 100°C; therefore, dicamba is relatively volatile. Dicamba is soluble in ethanol (92.2 g/100 ml), heavy aromatic naphthanes (5.2 g/100 ml), and xylene (7.8 g/100 ml). Water solubility of the acid is 0.45 g/100 ml (4,500 ppm) and that of the dimethylamine salt is greater than 72 g/100 ml (720,000 ppm).

Action in Vegetation

Broadleaf herbaceous plants (annuals and perennials) and woody plants (shrubs, hardwoods, and conifers) are sensitive to dicamba. When applied with 2,4-D or when the formulation includes 2,4-D as an active ingredient, the application rate of dicamba is one-half that of 2,4-D. In addition to these formulations designated for use on noncropland, dicamba is an effective post-emergence herbicide against broadleaf herbaceous weeds in lawns and turf, established grass crops, winter wheat, fall barley, oats and wheat, and field corn (Ashton and Crafts 1973).

Dicamba is readily absorbed by leaves and roots and is readily translocated throughout treated plants (Ashton and Crafts 1973). It concentrates in metabolically active parts of plants. Dicamba degradation rates in higher plants

vary greatly with species. In purple nutsedge, no metabolism of dicamba was detected after 10 days (Magalhaes et al, 1968; Ray and Wilcox 1969), but in wheat, dicamba was completely metabolized within 18 days although some persisted as a conjugate after 29 days (Broadhurst et al. 1966). Hydroxybenzoic acids (free or conjugated with normal plant constituents) are the primary degradation products. Their formation is responsible for termination of action of dicamba in treated plants. The relative amounts of dicamba and metabolites recovered from treated plants is concentration, time, and species dependent. Toxic effects of dicamba are related to its growth-regulating properties which are similar to those of 2,4-D. Rogerson and Foy (1968) reported that dicamba caused proliferative growth and a consequent increase in diameter of stems. Stem swelling was associated with decreased amounts of anthocyanin, a plant pigment. The mode of action of dicamba and other benzoic acid herbicides is discussed by Ashton and Crafts (1973).

Utilization by BPA

Dicamba (Banvel) is used by BPA in noncrop areas on rights-of-way for the management of weeds and brush beneath transmission lines and towers. BPA used on the average the equivalent of 14,628 pounds of dicamba (active ingredient) per year since 1978, and proposes to use 11,950 pounds of dicamba in 1983.

Banvel 4-O.S. is diluted with an oil solution of 2,4-D and applied with portable sprayers for basal-stem treatment to the point of runoff.

Banvel 5G is a pellet formulation applied with portable spreaders to the soil surface beneath shrubs and trees. Application is made just prior to or early in the rainy season and results in an effective application rate of dicamba (active ingredient) between 448 and 896 mg/m^2.

Banvel 4-W.S. and Banvel 720 are broadcast by air or ground equipment after leaves are fully developed and until three weeks before frost. Banvel 4-W.S. may be diluted with an aqueous 2,4-D solution before broadcast whereas Banvel 720 includes 2,4-D as an active ingredient. Dilution and application rates provide an effective application rate of dicamba between 112 and 336 mg/m^2. Banvel 4-W.S. and

Banvel 720 are also diluted with an equal quantity of water and applied to cut surfaces at any time of the year. Application of 0.5 to 1 ml of solution is made to each notch or injection point.

Chemical Fate and Distribution in the Environment

Dicamba does not persist in the environment due to physical, chemical, and biological processes. The most important physical characteristic of dicamba is its high water solubility which results in high soil mobility.

Soil

Dicamba is leached from surface soils and is one of the most mobile herbicides after it enters soil (Velsicol 1981). Soil mobility has been studied by Harris (1967) and Weber and Best (1971). Dicamba moves laterally, upwardly, and downwardly in the soil based upon studies of dicamba movement in runoff water (Trichel et al. 1968) and in leacheate of soil columns (Harris 1967).

A study of the fate of dicamba in a field lysimeter was conducted by Glass and Edwards (1979). Following application of 5.6 kg/ha, runoff and percolation water and soil samples were analyzed. Soil concentrations declined from 150 ppm to less than 1 ppm during the 11-month observation period. After 11 months, 1 ppb was detected in percolate water at a depth of 2.4 meters. It was concluded that loss of dicamba in runoff and percolate water would not likely pollute groundwater.

Steward and Gaul (1977) measured dicamba in soil following application of the amine salt at rates of 1.1, 2.2, and 4.5 kg/ha. Soil residues were concentration-dependent. After 42 days 0.02-0.12 ppm were present at soil depths of 10-20 cm but no dicamba was detected at 20-30 cm. Residues from trace amounts to 0.03 ppm were present at 265 days in the 10-20 cm zone. Only trace amounts were detectable in all soil samples after 385 days.

Microflora are important in dicamba degradation in soil. Smith (1973) estimated that four weeks was required for degradation of 50 percent of dicamba residues (half-life) in soil.

Alton and Stritzke (1973) reported that the dicamba half-life in various soils ranged between 17 and 32 days.

Water

Norris and Montgomery (1975) measured dicamba residues in streams after forest spraying in the Northwest. They concluded that there was low probability of the herbicide entering streams in overland runoff except during the first intensive storms after dicamba application.

Air

Application of dicamba sprays through the air results in the formation of aerosols whose fate and properties will be determined by their inherent physical characteristics. Dicamba volatility also is a source of the herbicide in air. Other reports include Eggemeyer (1971) and Wax et al. (1969). Burnside and Lavy (1966) reported extensive damage to soybeans from particle drift resulting from use of dicamba in post-emergence spraying of corn.

Vapors derived from foliar herbicide application to corn caused phytotoxicity in soybeans for three days following application in field experiments (Behrens and Lueschen 1979). Dicamba vapors cause phytotoxicity to soybeans when 60 meters downwind from treated corn. Volatility was reduced at lower temperatures and higher relative humidities in supporting laboratory studies. The volatile phytotoxic component of dicamba formulations was free of dicamba acid. The chemical fate of dicamba in air has not been studied.

Chemical Toxicology in Animals and Humans

Dicamba was introduced as a commercial product in the 1940s. It has been extensively used as a herbicide; however, very little toxicological data have been published. The majority of data summarized here are provided by an untitled document provided by the manufacturer (Velsicol 1981), as reviewed by Newton and Dost (1981).

Acute Effects

The rat oral LD50 for the acid is 2,900 $\pm$ 800 mg/kg body weight (WSSA 1979). One measurement in mice indicates an LD50 of greater than 4,600 mg/kg. The rat oral LD50 for the dimethylamine salt is 1,028 mg/kg (WSSA 1979). Based upon these estimates, dicamba would be classed as "slightly toxic".

Research with rabbits indicates that skin irritation occurs with extended contact. At all dosages (up to 2,500 mg/kg/day), no other signs of toxicity were observed. There was excessive mortality (7 of 32 rabbits) related to nontreatment-related causes. Dermal sensitization was produced following a three-week exposure period in which dicamba was applied three times per week. Animals were challenged (tested for skin sensitivity) 2 weeks after the last dose and again 48 hours later. Dicamba was considered to have moderate sensitization potential.

Acute toxicities of inhaled dicamba dusts are variable. Banvel (technical) has an LD50 (4-hour dust) of greater than 9.6 mg/liter. Banvel D (technical) has an LC50 (4-hour dust) of greater than 200 mg/liter. The basis for the discrepancy between the LC50s is not known.

In one method of interpreting toxicity data, it is presumed that acute (LD50) toxic doses for rat populations are similar to that for human populations. Given respective LD50s of 2900 and 1028 mg/kg for rat populations, it is presumed that the LD50 dose of dicamba acid and dicamba dimethylamine salt (active ingredient) for a population of 165-pound humans would be 7.7 ounces (0.046 oz/lb) and 2.7 ounces (0.016 oz/lb), respectively.

Chronic Effects

A 3-week study in which dietary concentrations of 658-23,500 ppm dicamba were fed to rats (1 of each sex per dose level) produced no evidence of toxicity in any of the animals.

A 90-day study in rats and dogs was conducted using a principal metabolite of dicamba, 2-methoxy-5-hydroxy-3,6-dichlorobenzoic acid. At 100 and 250 ppm in the diet no chemical, behavioral or other evidence of toxicity was observed.

Dietary levels of 5,000, 7,500, 10,000, 12,500 and 15,000 ppm were fed to rats (5 of each sex per dose level) for four weeks. Weight gain was decreased (to 40 percent) at the higher dietary levels. Seven of 10 of the rats at the highest dose and one at 12,500 ppm developed posterior weakness.

A "no effect" dietary level of 5,000 ppm was suggested in rats as a result of a 13-week study in which 10,000 ppm dicamba (technical) produced histological changes in liver. At the higher dose, the number of cytoplasmic vacuoles was reduced in liver parenchyma.

Two-year feeding studies were conducted in rats and beagle dogs. The rats were fed 500 ppm in the diet and the dogs were fed 50 ppm in the diet. Incidence and time of first appearance of tumors were the same in control and treated groups. A study of the carcinogenic potential of dicamba was also conducted on mice. Mice were given 100, 1,000, and 10,000 ppm in their diet during an intended 24-month test period. There was increased mortality at the highest test level, and the high-dose study was terminated at 14 months. The remainder of the study was terminated after 19.5 months rather than continuing to 24 months. The Velsicol summary does not indicate why the study was shortened. At the highest dose, decreased body weight and increased liver weight were reported. The only dose-dependent effect noted in the summary was an enlargement of liver cells. Whether the effect was reversible was not determined. No evidence of carcinogenicity was reported.

The two-year study with rats also addressed the issue of whether dicamba altered reproduction. Within the group of rats fed 500 ppm dicamba, two males and four females were mated. Also two treated males and four treated females were mated with untreated rats. No effects on reproduction were observed. A three-generation reproduction study at dietary levels up to 500 ppm using the second litter of each generation indicated no change in fertility, gestation, viability, or lactation in any group.

Teratogenicity studies were conducted in rabbits. They were given 1, 3, 10, or 20 mg technical dicamba/kg/day on days 6-18 of gestation. Post-implantation losses and a decreased number of live fetuses were recorded at 10 and 20 mg/kg/day. There was no effect at 3 mg/kg/day. A nontreatment-related decrease in post-implantation loss was observed in the 1/mg/kg/day group. No evidence of teratogenicity was obtained at 10 mg/kg/day or the lower doses. Fetal weight was decreased and post-implantation losses were observed at 10 mg/kg/day. This dose was considered nonteratogenic.

The results of the above study are the basis for the reproductive no-effect level for dicamba (3 mg/kg/day) used by Dost (1983) in a worst-case analysis of environmental herbicide exposure.

Tests of the mutagenic potential of dicamba have produced negative results. A dominant lethal test in male mice treated with either 1,000 mg/dicamba/kg orally or 30 mg/kg intraperitoneally caused no early embryonic death following weekly matings with fertile females.

Recombination assays with bacteria (Bacillus subtillus) exposed to dicamba concentrations of 1 mg/ml were negative. Salmonella (a bacterium) and Saccharomyces (a yeast) reversion assays with and without liver enzyme activation were negative up to 0.500 mg dicamba per plate, the highest concentration used. A negative host mediated assay (300 mg/kg, route not specified) was also reported in the Velsicol (1981) summary.

Dicamba was one of 18 chemicals tested for mutagenicity and its effect on unscheduled DNA synthesis as part of an EPA substitute pesticide program. Five short-term tests were performed. The microbial assay systems included: (1) the histidine reverse mutation in five strains of Salmonella typhimurium (TA 1535, TA 1537, TA 1538, TA 98, and TA 100); (2) the tryptophan mutation system in Escherichia coli WP-2 (a bacterium); (3) mitotic recombination in Saccharomyces cerevisiae D3; (4) unscheduled DNA synthesis in human fibroblasts (WI-38); and (5) relative toxicity assays in DNA repair-proficient and -deficient strains of E. coli (W3110 and P3478) and Bacillus subtillus (H17 and M45). A metabolic activation system prepared from Arochlor 1254 pretreated rats was used in all assays except in the relative toxicity tests. Dicamba was negative in each of the tests except for relative toxicity. On this basis dicamba is not considered mutagenic.

Potential Impact on Nontarget Organisms

Vegetation

Spray aerosol and vapor drift of dicamba to nontarget vegetation can have harmful effects. Sensitivity of plants to dicamba injury varies considerably.

Patric and Campbell (1970) classified West Virginia plants (number of species in parentheses) as: least susceptible (10); intermediate in susceptibility (16); and most susceptible (16). Dicamba pellets (Banvel XP) were less potent than liquid sprays applied directly to foliage. Maximum effectiveness of pellets was obtained during periods of rapid plant growth and high soil moisture.

Neel (1976) recorded the susceptibility of 23 species of "containerized environmental plants" to dicamba applied at rates of 2 and 4 pounds per acre. Phytotoxicity was observed in 17 species at 2 pounds per acre and in 16 species at 4 pounds per acre following root uptake of dicamba. Leaf distortion and epinasty (more rapid growth on

upper surface of plant tissue causing it to bend downward) were also observed. Plants were not killed at these application rates. Application of dicamba above root zone of sensitive species will cause phytotoxicity resulting from root uptake of the herbicide.

Fish and Aquatic Organisms (WSSA 1979)

Acute toxicity tests have been conducted with rainbow trout and bluegill. At 24, 48, and 96 hours, the LC50s in rainbow trout were 35, 35, and 28 ppm (mg dicamba/liter water). At 24 and 96 hours, the LC50s in bluegills were 130 and 23 ppm, respectively.

Further toxicity testing in small carp showed that the LC50 for the dimethylamine salt of dicamba was 659 ppm at 24 hours and 465 ppm at 48 hours. The median tolerance limits for dicamba in juvenile coho salmon were 151 and 121 ppm at 24 and 48 hours, respectively.

Wildlife (WSSA 1979)

The acute oral toxicity of the dimethylamine dicamba (LD50) in rabbits and pheasants is 566 mg/kg and 800 mg/kg, respectively.

Livestock

Metabolic studies have shown that dicamba and its metabolites are rapidly cleared by dairy cows (St. John and Lisk 1969; Oehler and Ivie 1980). Dosages of 2.2 mg dicamba/kg (or about 60 ppm of dietary dicamba) were administered for five days by Oehler and Ivie (1980). Within six hours of the last treatment, 89 percent of the total administered dose was recovered in urine and less than 0.02 percent was recovered from milk. Low-level exposures of ruminants through contaminated feed, forage, or water will not result in appreciable retention of residues by edible tissues or their secretion into milk.

The lethality of dicamba (dimethylamine salt) in chickens (LD50 673 mg/kg) is similar to the acute toxicity in other vertebrate species (WSSA 1979).

Hazard Assessment for Current Use Practices

Dicamba is an effective herbicide for vegetation management of annual and perennial broadleaf weeds and woody plants on

rights-of-way and other noncroplands. Dicamba and 2,4-D are used in combination to control brush and weeds below transmission lines and towers.

Based upon toxicity testing in laboratory animals, dicamba is classed as slightly toxic and dicamba herbicide formulations carry the Human Hazard Signal Word "caution". No evidence of carcinogenic or mutagenic hazard has been obtained. Teratogenic hazard appears to be limited to higher, albeit sublethal doses.

Metabolic studies in animals including laboratory and domestic species have shown that dicamba is rapidly excreted and not stored in tissues. Rapid elimination reduces the possibility of chronic toxicity.

The environmental fate of dicamba has been evaluated under a variety of laboratory and field conditions. Dicamba is not a persistent environmental pollutant. Dicamba is highly water soluble and mobile in soil. Runoff water resulting from rainfall immediately after application will contain dicamba at toxicologically insignificant levels. The amounts in runoff decline with time.

Soil microorganisms are of primary importance in dicamba degradation. Some chemical degradation of dicamba occurs in soil. Dicamba is a relatively volatile herbicide, and therefore is formulated to minimize losses by this route and to maximize absorption and herbicidal effects.

The major hazards associated with use of dicamba result from mobility in groundwater, the potential drift of the herbicide to nontarget plants, and from the possible movement of dicamba into the root zone of nontarget plants. Although contamination of groundwater is unlikely, the probability of groundwater contamination can be minimized by not using dicamba in areas with high water table. Drift of the herbicide to nontarget plants can also be minimized by appropriate application techniques. The herbicide has a low order or toxicity to fish, wildlife, and livestock. Exposure of these organisms is unlikely. Use of dicamba as indicated on the pesticide label results in a low degree of hazard to personnel, the public and the environment.

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DICHLOBENIL

Chemical Identification

Dichlobenil is the common name for 2,6-dichlorobenzonitrile. Casaron G-4 (EPA Regulation No. 148-614) is an herbicide which contains dichlobenil and is manufactured by Thompson-Hayward Chemical Company.

Dichlobenil is a white crystalline solid with an aromatic odor. The melting point is 145-146°C, and the boiling point is 270°C. Dichlobenil is thermally stable and does not decompose in daylight. The vapor pressure of dichlobenil is 5.5×10^{-4} mm Hg at 20°C and 1.5×10^{-2} mm Hg at 50°C. Five to 10 percent solutions of dichlobenil may be made in a variety of organic solvents. The water solubility of dichlobenil is approximately 18 ppm and the solubility of technical dichlobenil is approximately 25 ppm.

Action in Vegetation

The herbicidal properties of aromatic nitriles were discovered in the late 1950s and early 1960s. Dichlobenil inhibits germination of seeds of annual and perennial grasses and broadleaf plants and kills weed seedlings before or just after emergence, but does not control fully emerged weeds. In agriculture, it is used to control annual weeds in orchards and in small fruits such as blueberries and raspberries. Dichlobenil is also used in aquatic weed control (Comes and Morrow 1971).

Dichlobenil acts primarily on metabolically active buds, growing points, and root tips. It causes gross disruption of plant tissue which shows as a burning of foliage. Blistered or necrotic (dead) spots may appear within 24 hours. Plants with waxy cuticles have only intermediate susceptibility to foliar applications of dichlobenil. Slow translocation of dichlobenil in exposed plants is not related to its herbicidal action.

Absorbed dichlobenil is volatilized and metabolized in plants. Metabolic studies in bean plants (Verloop and Nimmo 1969) have resulted in a degradation scheme in which aromatic hydroxylation and conjugation of the products with natural substrates are quantitatively most important. Hydrolysis is the source of minor amounts of 2,6-dichlorobenzoic acid formed in some plants. Swanson (1969) has reviewed the degradation of dichlobenil and other aromatic nitrile herbicides.

Utilization by BPA

Dichlobenil is used by BPA to control weeds in ornamental plantings at substations. BPA used on the average the equivalent of 135 pounds of dichlobenil (active ingredient) per year since 1978. None is proposed for use in 1983.

Casaron G-4 is applied to the soil surface with portable spreaders in winter and early spring. Application of the formulated herbicide is made at an effective application rate of dichlobenil (active ingredient) between 450 and 900 mg/m^2.

Chemical Fate and Distribution in the Environment

Soil

The incorporation of dichlobenil into the soil lengthens its period of herbicidal activity. Losses by leaching are minor. Slow microbiological degradation of dichlobenil reduces soil residues.

When dichlobenil is incorporated into soil, adsorption on lignin or humic substances accounts for extensive soil binding. As a result, extensive rainfall does not leach dichlobenil to a depth below 4 inches (Swanson 1969). Very little leaching of dichlobenil has been observed in soils in the field under a variety of climatic conditions (Barnsley and Rosher 1961; Sheets et al. 1968; Beynon and Wright 1972).

The movement of dichlobenil in runoff water from four soil types was studied by Bailey et al. (1974). The herbicide was surface applied and incorporated. Simulated high intensity rainfall (5 inches in 2 hours) commenced 1 hour after application.

Herbicide transport occurred as discrete particles and as soil adsorbate. The greatest amounts were transported in runoff but highest concentrations were found on sediments. During the 2-hour test period, 70-80 percent of the dichlobenil loss occurred in the runoff under these extreme test conditions.

Studies of dichlobenil under field and laboratory conditions (Beynon and Wright 1972; Verloop and Nimmo 1970) have demonstrated the formation of 2,6-dichlorobenzamide. Three other unknown products were also observed by Verloop and

Nimmo (1970). Soil sterilization by heat blocked the degradation process. The benzamide is more persistent and mobile in soil than dichlobenil but it lacks toxicity to either plants or animals.

Other studies under laboratory conditions using a variety of soil types have also identified 2,6-dichlorobenzamide as a degradation product. Dichlobenil degradation rates were inversely related to the soil:water partition coefficients and were reduced in the presence of high organic soil matter.

Water

Dichlobenil is relatively persistent in pond water and muds (review by Newbold 1975). Frank and Comes (1967) reported that dichlobenil persisted in muds for 160 days. Extremes of persistence in water are represented by the studies of Ogg (1972) and Cope et al. (1969) who measured residues up to 16 and 189 days, respectively. Formulations used in water for aquatic weed control are slow-release preparations designed to maximize phytotoxic effects.

Ten weeks after dichlobenil application to two unlined irrigation channels, the amount remaining on the soil was 6 percent of the initial application. About 4 months later, water was flushed through the channel. It contained 0.18 ppm dichlobenil and represented no hazard to crops.

Dichlobenil metabolism by microorganisms in pond water and sediment was studied by Miyazaki et al. (1975). More than 75 percent of dichlobenil added to water and sediment was volatilized from the test systems. Unknown metabolites and 2,6-dichlorobenzamide were formed. ^{14}Carbon dioxide was formed in follow-up studies utilizing a cell suspension of Arthrobacter sp. (a bacterium).

Selective enrichment of total bacteria in freshwater lakes was observed after aquatic weed control treatments with dichlobenil and several other herbicides (Camper and Shively 1974).

Air

Dichlobenil is a volatile herbicide which is readily lost from soils unless incorporated. Soil and foliage residues may be lost as a vapor, especially at elevated high temperatures. Studies of the fate of dichlobenil in air are lacking.

Chemical Toxicology in Animals and Humans

Acute Toxicity

Results of acute toxicity studies have been reviewed by Van Genderen and Van Esch (1968). The acute oral LD50s of dichlobenil in rats, rabbits, and mice were 4,500, 270, and 2,100 mg/kg, respectively. In rats, liver and kidney damage was reported. Liver damage also was observed in rabbits.

The acute dermal LD50 in albino rabbits is 1,350 mg/kg. The subacute 21-day dermal LD50 of dichlobenil in rabbits is 500 mg/kg/day, and the apparent no effect level is 100 mg/kg/day. No significant dermal toxicity was noted at any of the doses tested (WSSA 1979).

In one method of interpreting toxicity data, it is presumed that acute (LD50) toxic doses for rat populations are similar to that for human populations. Given an LD50 of 4,500 mg/kg for rat populations, it is presumed that the LD50 dose of dichlobenil (active ingredient) for a population of 165-pound humans would be 11.9 ounces (0.072 oz/lb).

Chronic Effects

Van Genderen and Van Esch (1968) have also summarized the results of short-term and long-term feeding studies.

Short-term tests were conducted in rats, pigs, and rabbits. Male and female rats were fed 12 weeks on diets containing 0, 50, 100, 200, 300 or 500 ppm dichlobenil. Two female pigs were fed, 0, 20, 50, or 100 ppm for 26 weeks. Male and female rabbits were fed 0, 10, 20, 50, 500, or 1,000 ppm for 12 weeks. Organ weights and histopathological studies were conducted on selected tissues. It was concluded that 50 ppm was the apparent dietary no effect level.

Subsequent 2-year feeding studies in rats used dietary levels of 0, 20, 50, and 100 ppm dichlobenil. Histopathological study was conducted on liver, kidneys, lung, heart, brain, leg muscle, pituitary, adrenals, gastrointestinal tract, pancreas, bladder, thyroid, testes, salivary glands, spleen, and uterus. At these dietary levels, no treatment-related histopathological effects, including the incidence of tumors (carcinogenicity), were observed.

Rats from the 2-year study were also used in reproduction studies. After 3 months of dichlobenil feeding, 10 pairs from each group were mated and the offspring (F_1 generation) were fed the same dietary level as their parents.

From these groups (F_1), some rats were used for other studies and others were bred to produce the F_2 generation. Reproduction efficiency was judged on the number of females with litters, the total number of young after 1, 5, and 21 days, and the body weight of the litter after 21 days. Growth, haematology and organ weights and histopathology at necropsy were studied in parents and the F_1 generation. Growth retardation was observed at 50 and 100 ppm dietary dichlobenil. More marked effects were seen in the F_1 generation. No haematological effects were observed. Some treatment-related increases in organ weights (liver, testes, kidney) were observed. The reviewers concluded that dichlobenil had no apparent effects on reproduction at dietary levels of 50 ppm or below.

A study of fetal abnormalities was conducted in mice given 60 mg dichlobenil/kg by the oral route on days 3-14 of pregnancy. The mice were killed on day 21. No differences between control and treated groups were observed with respect to total number of fetuses, litter size, or percentage of dead fetuses.

Potential Impact on Nontarget Vegetation

Vegetation

Use of dichlobenil granules in ornamental plantings results in control of weeds. Injury to viburnum, tallhedge, and buckthorn in Ohio nurseries resulted from application of 6 lb. dichlobenil/acre. Chlorosis along the leaf margins, browning, and stunting were reported. The plants recovered their former condition and no lasting effects (other than temporary growth setback) were observed (Smith 1972, cited in U.S. Forest Service 1981).

Use of dichlobenil in croplands also demonstrates the potential for phytotoxicity, perhaps resulting from accumulation of 2,6-dichlorobenzamide along leaf margins (Verloop 1972). Areas where there is rapid leaf transpiration may be more prone to this type of damage.

Fish and Aquatic Organisms

Acute toxicity testing in fish has included 24- and 48-hour LD50 determinations in pumpkinseed, bluegill, and largemouth bass. The LD50s ranged between 10 and 20 ppm.

Since dichlobenil is used in aquatic weed control, there have been opportunities to observe the responses of aquatic organisms to herbicidal concentrations of dichlobenil.

Boller et al. (1973) described the responses of goldfish to an application of dichlobenil to control surface growth of weeds. At the highest concentration (6.4 mg dichlobenil/liter [6.4 ppm]), fish body weight and abnormal behavior was observed. No fish deaths were reported. At lower herbicidal concentrations, no effects were observed.

The sensitivity of eight species of aquatic invertebrates has been established (reviewed by Bunting and Robertson 1975). Under laboratory conditions, the LC50s ranged from 3.7 ppm (48 hour) in Daphnia pulex (a water flea) to 34 ppm (48 hour) in Asellus brevicaudus (an isopod). The sensitivity of these invertebrates is similar to that of the fish that have been tested.

A field study was conducted by Cooke (1977) to evaluate the sensitivity of three amphibians to concentrations of dichlobenil used in aquatic weed control (mg/liter). Caged frogs, toad tadpoles, and uncaged smooth newts were observed. Tadpole development and activity were not affected and no mortality was observed. Tadpoles from the treated pond were heavier than controls after 18 and 32 days due to a bloom of a desirable algae. The caged frogs showed no toxic effects. Based upon longer term observations, the suitability of the pond as newt habitat was reduced by the herbicide treatment.

Wildlife

The dietary LC50s (8 day) for Japanese quail and ring-necked pheasant were 5,000 and 1,500, respectively (Hill et al. 1975).

Benyon and Wright (1972) reviewed the environmental fate of dichlobenil and concluded that terrestrial animals would be unlikely to contact appreciable amounts of the herbicide during or after application. Dichlobenil is rapidly eliminated from rats (Griffiths et al. 1966; Wit and Van Generen 1966), beagle dogs (Griffiths et al. 1966), and rabbits (Wit and Van Generen 1966) in urine. No tissues retain dichlobenil residues, thereby reducing the possibility of chronic toxicity.

Livestock

Dichlobenil is rapidly eliminated from dairy cows (St. John and Lisk 1967).

Hazard Assessment for Current Use Practice

Dichlobenil inhibits germination of seeds of both grasses and broadleaf plants, but it does not affect emerged plants. BPA makes very limited use of this herbicide, specifically in the control of weeds in ornamental plantings at substations.

Based upon toxicity testing in laboratory animals, dichlobenil has a low order of toxicity. As a result, CAUTION is the Human Hazard Signal Word displayed on the herbicide label.

Metabolic studies using laboratory rodents and domestic animals have shown that dichlobenil does not accumulate in tissues. No evidence of carcinogenic, teratogenic, or reproductive activity has been obtained in toxicity testing. Mutagenicity data are not available.

The environmental fate of dichlobenil has been evaluated under a variety of laboratory and field conditions. Due to its relatively high volatility, it is most effective when mixed with soil or leached into soil with rainfall or irrigation shortly after application. Dichlobenil is strongly adsorbed to soil which, combined with its low water solubility, keeps the herbicide in place for long periods. It may remain effective for a year or more.

Use of dichlobenil as indicated on the pesticide label results in a low degree of hazard to personnel, the public, and the environment. BPA makes very limited use of dichlobenil in ornamental plantings at substations.

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DIURON

Chemical Identification

Diuron is the common name for 3-(3,4-dichloro-phenyl)-1-dimethylurea. Karmex is the trade name of an herbicide containing diuron and used by BPA until recently. Diuron now is used by BPA only as formulated in Krovar I (EPA Reg. No. 352-352-AA). Diuron and bromacil are active ingredients in Krovar I. A separate Herbicide Background Statement has been prepared for bromacil (page A-34).

Diuron is a white, crystalline solid. It is odorless and melts at 158-159°C. Thermal decomposition occurs at 180-190°C. The vapor pressure of diuron is 0.31×10^{-5} mm Hg at 50°C and 148×10^{-5} mm Hg at 100°C. Diuron is chemically stable toward oxidation and moisture under normal conditions. Diuron hydrolysis is negligible at ordinary temperatures at neutral pH. Elevated temperatures and alkaline or acid conditions cause rapid hydrolysis. Small amounts of diuron are soluble in water and hydrocarbon solvents. The limit of water solubility is 42 ppm (Klingman and Ashton 1975).

Action in Vegetation

Many annual and perennial grasses and herbaceous weeds on noncropland areas are sensitive to diuron. Diuron is used as a preemergence treatment.

Diuron is readily absorbed by roots and rapidly translocated to upper plant parts. Applications to leaves are not translocated to untreated leaves to a significant extent (Ashton and Crafts 1973).

The primary toxic action of diuron in plants results from inhibition of photosynthesis (Ashton and Crafts 1973). The toxic effects of diuron are primarily observable in leaves. Leaves may merely show slight yellowing or chlorosis, which develops during a period of several days following exposure to the herbicide. Higher amounts produce leaves which appear water-soaked and become necrotic within a few days (Ashton and Crafts 1973).

Utilization by BPA

Diuron is used by BPA to control weeds at substations in noncrop area. BPA used the equivalent of 202 and 340 pounds of diuron (active ingredient) in 1980 and 1982, respectively, and proposes to use 230 pounds of diuron in 1983. Diuron was not used in 1978 and 1979. BPA is currently replacing Karmex (DuPont) with Krovar I (DuPont); 59 percent of the diuron applied in 1981 was formulated as Karmex, whereas only 18 percent of the diuron to be applied in 1982 will come from remaining Karmex supplies.

Krovar I is used at substations and applied to the soil surface in spring or summer. Portable sprayers direct an aqueous spray mixture at a rate providing an effective application rate of diuron (active ingredient) between 179 and 1,792 mg/m^2.

Chemical Fate and Distribution in the Environment

Soil

Urea-based herbicides such as diuron are relatively persistent in soils. Where complete control of vegetation has been obtained and with little or no leaching, diuron may persist for 24 months. The principle factors affecting persistence of diuron in soil are microorganism decomposition, leaching, adsorption on soil colloids, and photodecomposition. Photodecomposition is only important during the time the herbicide remains on the soil surface. Volatility and chemical decomposition are of minor importance in the reduction of soil diuron concentrations (Klingman and Ashton 1975).

Microorganism decomposition is most important to the reduction of soil diuron concentrations. Soil bacteria including *Pseudomonas* sp., *Xanthomonas* sp., *Sarcina* sp., and *Bacillus* sp. and fungi including *Penecillium* sp. and *Aspergillus* sp. can utilize diuron as a direct source of energy (Hill and McGahen 1955).

Conditions which favor microorganism decomposition include moderate temperatures, moderate moisture, and soil aeration. Extremes of cold, wetness, or dryness, therefore, cause extended persistence of diuron.

Soil adsorption is relatively more important than water solubility in determining the rate of leaching of urea-based herbicides (WSSA 1979). The water solubilities of diuron, monuron, and bromacil at 25°C are 42, 230 and 815 ppm,

respectively. Each of these herbicides is used in BPA vegetation management. Adsorption on Keyport silt loam was measured as the amount of urea herbicide present on soil in equilibrium with 1 ppm in soil solution at about 22°C. Diuron adsorption was 4.0-5.2 ppm. It was more extensively adsorbed than either monuron (2.6 ppm) or bromacil (1.5 ppm).

Liu et al. (1970) studied diuron persistence in soil under greenhouse and laboratory conditions. Thirteen Puerto Rican soils were used and oats were the bioassay plants. Using an agitated slurry technique, these workers measured the ratio of the amount of herbicide adsorbed to the amount in solution at equilibrium. Thirty-four soil types were studied. Adsorption of diuron highly correlated with soil organic matter and cation exchange capacity. Soil magnesium was also correlated with diuron adsorption.

Bowmer (1972) and Khan et al. (1976) have shown that diuron does not accumulate in orchard soils as a result of annual applications. Herbicide residues were generally confined to the upper 15 cm of soil. Degradation rates at any time were generally proportional to the herbicide concentration in the soil.

Diuron leaching in field lysimeters was studied by Liu (1974). Diuron leached to a maximum depth of 36 inches and the highest concentration was detected in water samples taken one week after application. Most extensive leaching occurred during week 1 and no leaching was measurable 16 weeks after application. The leaching loss of diuron was 3.6 percent of the total application.

Surface-applied diuron in the field did not move below 5 cm in a Monona silty clay loam which receive 20 cm of water during a 54-day test period (Majka and Lavy 1976). The movement of diuron in hand-packed soil columns in the laboratory was similar to movement in the field. Diuron broke down more readily at 35°C than at 5, 20, or 50°C in 20 weeks.

Research on the fate of diuron in soil has focused upon terrestrial use at rates of 2 pounds/acre or less. Under these conditions, minimal problems of chemical carryover occur.

Under most climatic and soil conditions, the rate of diuron disappearance will equal or exceed 80 percent per year, depending on soil type. The principal soil factor is organic content which is inversely correlated with persistence and positively correlated with the rate of microbial degradation (U.S. Forest Service 1981).

Water

Diuron has been evaluated as a broad-spectrum, aquatic herbicide (Johnson and Julin 1974). Factors affecting the efficacy of diuron in this use include light, temperature, and water quality. Adsorption by organic matter and clays also can influence phytotoxicity. Diuron is registered for aquatic use in three states and at least four foreign countries, although a Federal label has not been registered (Johnson and Julin 1974). Evaluation of this pattern of use of diuron will contribute to knowledge of effects on aquatic organisms that might result from accidental or unintentional introduction into water.

When used for aquatic weed control in drainage ditches, diuron is nonpersistent (Johnson and Julin 1974). Water concentrations were 10-20 percent of the initial concentration within 10 days after application. Most residual diuron was found in the upper 0-5 cm of bottom sludge. After three months residues were not detectable.

Diuron may be a contaminant of irrigation water as a result of its use at high application rates (30 pounds/acre) for aquatic weed control. Water samples taken from water at distances of 10 and 100 feet below the treated site contained 0.72 to 1.78 ppm and nondetectable to 0.93 ppm, respectively. Water taken 1,000 feet below the treated site did not contain detectable amounts of diuron.

Studies of estuaries receiving water from plantations treated with diuron showed soil and sediment residues of the herbicide (Green et al. 1977). After six months, sediment concentrations were generally 500 ppb (oven-dry basis). Higher concentrations were found in sites subject to local contamination from spray equipment loading areas.

Diuron is transported in solution rather than adsorbed to soil particles. Maximum diuron concentrations in runoff were measured following rainstorms that occurred soon ater application (Willis et al. 1975). In a three-year period, maximum seasonal losses were 0.12 percent. In this study, diuron dissipated primarily by microbial degradation. Together these factors resulted in a soil half-life of about two months at rates of application used in agriculture.

Chemical breakdown of diuron in natural surface water is a minor route of degradation in the aquatic environment.

Air

Losses from soil via volatility and photodecomposition are not significant except when diuron is exposed on the soil surface for several days or weeks under dry conditions (WSSA 1979). Demethylation of diuron may occur in sunlight (Crosby 1976) with resultant loss of phytotoxicity.

Chemical Toxicology in Animals and Humans

Acute Effects

Diuron has a low order of acute oral toxicity in the rat. The LD50 (14-day) for male rats was 3,400 mg/kg body weight.

A 50 percent water paste of diuron was not irritating to intact skin of guinea pigs. Moderate irritation occurred on broken skin. A 10 percent suspension in water caused mild irritation. Diuron did not produce allergic skin sensitization.

Eye tests with Karmex weed killer (wettable powder) in rabbits produced very mild conjunctival irritaion. No effects on cornea or iris were observed.

In one method of interpreting toxicity data, it is presumed that acute (LD50) toxic doses for rat populations are similar to that for human populations. Given an LD50 of 3,400 mg/kg for rat populations, it is presumed that the LD50 dose of diuron (active ingredient) for a population of 165-pound humans would be 9.0 ounces (0.055 oz/lb).

Chronic Effects

A 90-day feeding study in rats disclosed no growth depression at a dietary level of 400 ppm or less. Slight growth depression occurred in males at 2,000 ppm and the effect was marked in both sexes at 2,500 ppm and higher. Slight anemia and enhanced erythropoiesis (red blood cell production) were observed at 250 ppm in females and in both sexes at 2,500 ppm and above. An abnormal blood pigment, sulphaemoglobin, was detected at the higher feeding levels (2,000 ppm and higher).

Feeding studies using two dogs revealed no effects at 160 ppm in diet for one month. Slight growth retardation and moderate anemia were induced by two-month feedings of increased dietary levels commencing at 400 ppm and terminating at 2,400 ppm.

Two-year feeding studies were conducted in rats and dogs at dietary levels of 0, 25, 125, 250, and 2,500 ppm. No effect was seen at 25, 125 and 250 ppm in either species, with the exceptions of a trace of abnormal blood pigment in some animals at 125 and 250 ppm diuron and a trend toward reduced red blood cell counts in dogs at 250 ppm. At the highest concentration (2,500 ppm), the principal findings in dogs and rats were growth retardation, slight anemia, presence of abnormal blood pigment, enlargement of the spleen (occasionally in rats) and liver (dogs) and increased erythropoiesis with splenic haemosiderosis (a symptom of elevated levels of iron). No histological changes were seen in the brain, lungs, heart, kidney, urinary bladder, adrenal, gonads, stomach, small and large intestine, muscle, and thyroid gland.

There was no evidence in these studies that diuron was carcinogenic.

A three-generation reproduction study in rats maintained on 0 and 125 ppm diuron yielded no evidence of harmful effects.

Metabolic studies using dogs and rats fed 25-2,500 ppm diuron for nine months to two years showed tissue concentrations to be proportional to dietary intake. No evidence of tissue storage was obtained. Diuron was excreted in urine and feces. Diuron, demethylated derivatives, and 3,4-dichloroaniline, and 3,4-dichlorophenol were detected.

The above findings were reported by Hodge et al. (1967). This report is the most extensive compilation of diuron toxicity data in the literature.

Two additional studies on carcinogenicity are available. Innes et al. (1969) used maximum tolerated doses in mice in research conducted by Bionetics Research Laboratories and the National Cancer Institute. Diuron was among 89 compounds which gave no indication of tumorigenicity after oral administration for 78 weeks. Rubenchik et al. (1973) studied the relationship between chemical structure and carcinogenic activity of urea derivatives including diuron. Histologic examination of the various organs of the 30 rats necropsied (of 40 animals at the start) showed 20 to have tumors in various organs, including the liver (14), stomach (13), lungs (3), intestine (6), pancreas (6), and kidney (3). The diet level in this study (4,500-9,000 ppm) and the duration of the observation period (116 weeks) both exceeded those used in the studies of Hodge et al. (1967). The results of the carcinogenicity investigations of Hodge et al. (1967) and Innes et al. (1969) contrast with those of Rubenchik et al. (1973). Diuron is not classed as a carcinogenic herbicide by the U.S. Environmental Protection Agency.

Studies of the teratogenic potency of diuron have been conducted (Mrak 1969; Khera et al. 1979). No significant increase in anomalies in litters of two strains of mice were observed following administration of 215 mg diuron/kg in large-scale screening studies by Bionetics Research Laboratories under a contract for the National Cancer Institute (Mrak 1969). Karmex at 250 and 500 mg/kg dosages once daily from the 6th to the 15th day of gestation "manifested an increased incidence of malformed fetuses or maternal toxicity" in rats. The lowest dosage (125 mg/kg) caused a single anomaly of delayed ossification of the calvarium (upper skull) which was not dose-dependent and was of borderline statistical significance (Khera et al. 1979).

Potential Impact on Nontarget Organisms

Vegetation

To evaluate the effects of diuron on crops, numerous studies of persistence and residual toxicity (carryover) have been conducted. Lukin (1970) applied diuron to potato plantings and concluded that when effective weed control was obtained, no effect on the culinary and food qualities of the crop was evident. Boger and Schlue (1976) reported that diuron did not cause effects on photosynthesis in algae following long-term exposures. Nitrate and nitrite metabolism and protein synthesis were not correlated with the sensitivity of four plants to foliar diuron treatments (Santakumore and Roma Das 1977).

Fish and Aquatic Organisms

An extensive review of literature and other data on diuron toxicity was published by Johnson and Julin (1974). The lethal concentrations of diuron to 50 percent of test populations (LC50) were determined for 12 species of fish including rainbow trout (Salmo gairdneri), lake trout (Salvelinus namaycush), and coho salmon (Oncorhynchus kisutch). The LC50s ranged from 1.2 to greater than 32 ppm for test periods from 24 to 96 hours in the 12 species reported. The 96-hour LC50s in the rainbow trout, lake trout, and coho salmon were 3.5, 2.5, and 1-10 ppm, respectively.

The acute toxicity of diuron was not significantly altered by water pH and hardness. Increased temperatures increased the toxicity of diuron to bluegills (Lepomis macrochirus) (LC50 9.1 ppm at 7°C, 2.1 ppm at 30°C).

Concerning use of diuron in aquatic weed control, Johnson and Julin (1974) stated: "Relatively few reports indicate that diuron is acutely toxic to fish in ponds treated at recommended levels. On the contrary, most workers have concluded that diuron is relatively nontoxic at these levels and mortalities are more often due to indirect effects such as oxygen depletion." Diuron is sometimes used in fish hatcheries for aquatic weed control. Concerns remain about possible chronic toxicity since some workers have reported decreased rates of growth (Johnson and Julin 1974).

Other studies reviewed by Johnson and Julin (1974) noted pathological effects on blood, liver, gill, muscle, gall-bladder, pancreas, spleen, gonads, kidney, brain, and heart in controlled laboratory studies. The potential toxicity of diuron to fish has been extensively documented in the literature.

The toxicity of diuron to freshwater and marine invertebrates has been reviewed by Johnson and Julin (1974) in conjunction with their work with fish. Estimates of toxic or lethal levels in 13 species of freshwater invertebrates and three species of marine invertebrates were made. Toxic concentrations were below or within one order of magnitude of the solubility limit (42 ppm) in all but one case. The 48-hour LC50 for diuron in crayfish was 800 ppm. Diuron toxicity to invertebrates is of a magnitude similar to that in fish.

Wildlife

Feeding studies in wildlife are used to assess toxicity to several indicator species. Results of these studies are summarized in a Technical Data Sheet (Du Pont 1980). The eight-day dietary LC50s for diuron were determined in bobwhite quail, Japanese quail, ring-neck pheasant chicks, and mallard ducklings. The LC50 for bobwhite quail was 1,730 ppm. In the other three cases, the LC50 was greater than 5,000 ppm. No evidence of exceptional toxicity to wildlife relative to that in laboratory rodents was obtained.

Livestock

Cattle and sheep were dosed by either drench or capsule and chickens by capsule only. Signs of poisoning in cattle and sheep were anorexia, depression, dyspnea (shortness of breath), and prostration. Uncoordinated gait was observed in poisoned sheep. Chickens showed weight loss, and necropsy revealed congestion of the intestinal mucosa and an enlarged, congested liver.

Feed contaminated by diuron could be toxic to livestock. Rates in excess of one pound/acre would be hazardous to chickens. Rates up to 9.6 pounds/acre would not be hazardous for cattle and sheep (Palmer and Radeleff 1969).

Hazard Assessment for Current Use Practice

Herbicides containing diuron are used to a very limited extent by BPA. Karmex was used to control weeds along streets and sidewalks at BPA facilities. Krovar (which contains bromacil and diuron) is used at substations as a broadcast or directed spray applied using portable sprayers.

Diuron is effective for the control of grasses and broadleaf weeds. Diuron is applied to the soil surface and to newly emerged, rapidly growing weeds.

Based upon testing in laboratory animals, diuron has a low order of toxicity. As a result, CAUTION is the Human Hazard Signal Word displayed on the herbicide label (see Toxicology Overview).

Metabolic studies in microorganisms, plants, laboratory rodents, and domestic animals have revealed that diuron is extensively metabolized and is not retained in tissues. No evidence of mutagenic activity has been reported.

Diuron has been shown by Russian investigators to have carcinogenic activity in rats. Two studies in rodents and the long-term feeding study in dogs did not demonstrate that diuron caused excess tumors. The discrepancies between results of these studies have not been resolved.

No epidemiological studies have been performed to assess the extent to human exposure associated with the use of diuron. The extent of exposure can be reduced by good hygiene. The extremely small scale of diuron usage by BPA further minimizes any real or apparent threat to health.

The environmental fate of diuron has been studied under a variety of laboratory and field conditions. Microbial degradation is the primary factor in the disappearance of diuron from soils. The extent of soil adsorption increases as clay and/or organic matter increases. Leaching is not an important route of loss except in sandy soils. Losses by volatilization and photodecomposition are negligible. Diuron is sufficiently persistent that phytotoxicity will be retained at least one season, and longer at higher rates or in dry climates.

Based on available evidence, the low acute toxicity, and the degradation of diuron under environmental conditions, use of diuron as indicated on the pesticide label results in a low degree of hazard to personnel, the public, and the environment.

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GLYPHOSATE

Chemical Identification

Glyphosate is the common name for N-(phosphonomethyl)glycine. Roundup is the trade name of the herbicidal formulation of the isopropylamine salt of glyphosate manufactured by Monsanto Company. The EPA registration number is 524-308-AA.

Glyphosate is a white,odorless solid which melts at 200°C. It has a "negligible" vapor pressure (Monsanto, 1980) and the pure material has a density of 0.5 g/cm^3. The solubility of glyphosate is 1.2 percent (12,000 ppm) in water at 25°C.

Roundup is a viscous, golden yellow liquid which has a specific gravity of 1.17 gm/ml and is completely miscible with water. The formulation is chemically stable and has a shelflife of at least 4 years under warehouse conditions (Monsanto 1980).

Action in Vegetation

Glyphosate is a broad-spectrum, nonselective herbicide applied as a solution in water to the foliage of target plants. Glyphosate is effective on deep-rooted perennial species and on annual and biennial species of grasses, sedges, and broadleaf weed (WSSA 1979).

In addition to its use in vegetation management on non-croplands, glyphosate is used extensively in agriculture. Tolerances (i.e., maximum permissible residue levels) are established in over 50 feed or food crops.

Baird et al. (1971) introduced glyphosate as a broad-spectrum, post-emergence herbicide. Glyphosate is readily absorbed by plant foliage and is translocated to underground roots or rhizomes; it is a strong inhibitor of sprouting by perennial species. Control of most annual weeds is complete at low rates of application (0.3-1.0 pound/acre).

Phytotoxicity is slowly evident and, depending upon species, may not appear for 2-4 days for annual species and after 7-10 days or longer for perennials. Visible signs of toxicity include wilting and yellowing of the plant which advances to complete browning and deterioration of plant tissue (Monsanto 1973).

The mechanism of action of glyphosate is not known. The herbicide inhibits aromatic amino acid biosynthesis (Jaworski 1972). Further studies have shown that glyphosate inhibits synthesis of essential amino acids (Shaner and Lyon 1980) and promotes destruction of photosynthetically active pigments in foliage (Hoagland 1980). Studies using ^{14}C-radiolabelled glyphosate have shown that the herbicide is not metabolized in plants.

Utilization by BPA

Glyphosate (Roundup) is used to a limited extent by BPA to control weeds and brush beneath transmission lines and towers, around poles, and at substations. Portable sprayers are used to direct spray to foliage of target plants late in the growing season or after target plants have reached maturity. Roundup is diluted with water (10 quarts per 100 gallons), resulting in an effective glyphosate concentration of 25,000 ppm. Plant foliage is sprayed until wet, but not to the point of runoff. BPA recomends an effective application rate of about 30 gallons diluted solution/acre (140 mg/m^2). BPA used on the average the equivalent of 450 pounds of glyphosate (active ingredient) per year since 1978, and proposes to use 12 pounds of glyphosate (active ingredient) in 1983.

Chemical Fate and Distribution in the Environment

Soil

Glyphosate is very strongly adsorbed by soil; as a result its phytotoxicity is low following soil application. Accordingly, glyphosate leaching is also very low (WSSA 1979).

The initial rapid inactivation of glyphosate in the soil results from adsorption on soil particles (Hance 1976). Further inactivation is due to microbial breakdown. Glyphosate itself does not sustain microbial growth but soil bacteria are responsible for extensive glyphosate degradation (Sprankle et al. 1975). Rueppel et al. (1977) also concluded that physical adsorption was more important than chemical breakdown in the degradation of glyphosate.

Water

The fate of glyphosate in water was studied by Comes et al. (1976). Glyphosate was applied at high rates (5.6 kg/ha) to ditch banks of dry canals. Neither glyphosate nor its major

soil metabolite, aminomethylphosphonic acid, were present in the first flow of water 5-6 months later.

Newton and Dost (1981) studied the pattern of glyphosate behavior in water; concentrations in a forest stream diminished rapidly, partially through adsorption to bottom sediments where extensive microbial breakdown occurred. After aerial application of 3.3 kg/ha to an open stream, maximum concentrations were 0.2 ppm in a beaver pond, 0.08 ppm within 6 hours, and 0.005 ppm by day 3. They concluded that concentrations observed in streams were at no time high enough to cause injury to aquatic organisms.

Air

Glyphosate volatility is "negligible" (Monsanto 1980). Losses from soil and water due to volatilization are very small (Rueppel et al. 1977). The fate of aerosolized glyphosate has not been described.

Chemical Toxicology in Animals and Humans

Few published reports on glyphosate exist in the archival scientific literature. Safety-hazard assessment studies on Roundup have been prepared and filed with the U. S. Environmental Protection Agency as required by the Federal Insecticide, Fungicide, and Rodenticide Act. Since the substance of these reports is proprietary information, the following information is based upon summaries provided by the manufacturer and a recent review by Newton and Dost (1981). The most recent Roundup Herbicide Bulletin (Monsanto 1982) also reviews health and environmental studies conducted by Monsanto Co.

Acute Effects

Glyphosate and Roundup have a low order of acute toxicity. Their acute oral LD50s in rats are 5,600 mg glyphosate/kg and 5,400 mg Roundup/kg. The acute oral LD50 is 3,800 mg glyphosate/kg in rabbits. Glyphosate and Roundup also have low toxicity following dermal exposure. The dermal LD50 in rabbits for each material is greater than 5,000 mg/kg.

Glyphosate is slightly irritating to the rabbit eye (6.9 on a scale of 0 to 110.0). Roundup is moderately irritating to the rabbit eye based upon a battery of studies. Primary

skin irritation testing in rabbits revealed that glyphosate was nonirritating and that Roundup was moderately irritating to rabbit skin. Rats exposed for 4 hours to air containing 12.2 mg Roundup/liter survived and showed no abnormal reactions. Those rats were necropsied 10 days later. No treatment-related gross pathology was noted.

In one method of interpreting toxicity data, it is presumed that acute (LD50) toxic doses for rat populations are similar to that for human populations. Given an LD50 of 5,600 mg/kg for rat populations, it is presumed that the LD50 dose of glyphosate (active ingredient) for a population of 165-pound humans would be 14.8 ounces (0.090 oz/lb).

Chronic Effects

Longer term studies have been conducted in rats and dogs given different levels of technical glyphosate. "Technical" indicates that the chemical was in the form manufactured for sale. Ninety-day feeding studies in rats used dietary levels of 200, 600, and 2,000 ppm. Controls and treated rats did not differ with respect to mean body weight, food consumption, behavioral reactions, mortality, hematology, blood chemistry, and urinalysis. At necropsy, no relevant gross or histopathologic changes were observed.

Two-year feeding studies using rats and dogs at dietary levels of 30, 100, and 300 ppm indicated no effect levels of 100 ppm and 300 ppm in the rat and dog, respectively. A three-generation rat reproduction study was negative at 300 ppm. Two teratology studies in rats were negative at 30 mg/kg/day (the highest dose tested) (Newton and Dost 1981). This result is the basis for the reproductive no-effect level for glyphosate (30 mg/kg/day) used by Dost (1983) in a worst-case analysis of environmental herbicide exposures.

The herbicide bulletin (Monsanto 1980) contains the following information concerning glyphosate metabolism and potential for bioaccumulation: "Metabolism test results show that glyphosate does not accumulate in animals, birds and aquatic species, and thus will not be passed up the food chain. The lack of accumulation is also supported by the high water solubility of Roundup herbicide and rapid depletion of glyphosate from the body. In fact, when milk from lactating cows and eggs from chickens fed diets with glyphosate were analyzed, residues were not detectable (less than 0.025 ppm parent and metabolite)."

Newton and Dost (1981) report that EPA has indicated that "additional oncological studies" with glyphosate are desirable, but that available lifetime studies indicate that glyphosate has low oncogenic potential. EPA has set a human acceptable daily intake standard of 50 mg glyphosate/kg/day.

The results of two additional inquiries concerning glyphosate toxicology are also summarized by Newton and Dost (1981). EPA has concluded that there is neither consumer nor applicator hazard related to N-nitrosoglyphosate which may be present as a glyphosate contaminant. Glyphosate is a phosphonoalkyl compound, similar in some respects to some insecticidal acetylcholinesterase inhibitors. The reviewers concluded that glyphosate was not an anticholinesterase.

Although toxicity testing data remain proprietary, a review of all available information has been conducted by F. N. Dost and J. M. Witt of Oregon State University. The reviewers had access to every document pertinent to a hazard assessment and were satisfied in all respects that prescribed use of Roundup represents no human health hazards (Newton and Dost 1981).

No cases of poisoning in humans have been observed or reported (WSSA 1979).

Potential Impact on Nontarget Organisms

Vegetation

Studies of the fate of glyphosate in forest foliage have been conducted by the Forest Research Laboratory at Oregon State University; a summary of results was prepared by Newton and Dost (1981). As applied in forestry, an application rate of 3.3 kg glyphosate/ha (2.9 lb/ac) results in a level of about 300 ppm on overstory foliage (i.e., 100 ppm/kg/ha applied). This application rate results in 20-200 ppm at the top of the browse layer (i.e., the height deer and elk can reach on shrubs and trees). Understory shrub canopies can be expected to have approximately one-third the level of overstory foliage.

After application, glyphosate degrades or otherwise disappears from each layer of vegetation rapidly. Half-lives in every component of vegetation were less than 2 weeks and were as little as one day in overstory foliage (Newton and Dost 1981). The possibility of phytotoxicity from residual amounts of glyphosate in soil is unlikely due to rapid and

tenacious soil adsorption. Soil-adsorbed glyphosate is unavailable for root absorption; similarly, residues are not available to affect germinating plant material.

Glyphosate has been extensively evaluated for use in agriculture, ornamental and landscape plantings, and wildland management of noxious weeds. The evaluation related to forest management is less comprehensive. These evaluations confirm that glyphosate is an extremely broad-spectrum herbicide, and they provide guidance for the selective use of glyphosate in vegetation management. Lund-Hoie (1976) found glyphosate sensitivity among all common brush and broadleaf species that were abundant in Norway forest plantations.

Newton (1978) demonstrated use of glyphosate to control deciduous brush in Oregon. Salmonberry, cascara, bitter cherry, ocean spray, thimbleberry, California hazelnut, red elderberry, and vine maple were susceptible to glyphosate at application rates of 0.56-1.12 lbs/acre (0.63-1.25 kg/ha). Control of red alder, bigleaf maple, and poison oak was variable at rates up to 1.5 lbs/acre (1.68 kg/ha). Herbaceous vegetation in active growth was sensitive to the phytotoxicity of glyphosate. Douglas-fir, noble fir, grand fir, and sitka spruce were not affected except for minor tip injury at rates of application up to 0.75 lbs/acre (0.84 kg/ha). Dieback of 3-12 inches of terminal occurred at 1.5 lbs/acre (1.68 kg/ha). Evergreen shrubs (e.g., greenleaf manzanita, Pacific madrone, salal, and evergreen huckleberry) were not severely affected at rates of glyphosate which caused damage to conifers.

At an applied rate of 3.3 kg/ha (2.9 lb/ac), glyphosate suppresses nearly all vegetation receptive at time of application. Evergreen shrubs and trees, however, are highly tolerant (Newton and Dost 1981).

Glyphosate gave 100 percent control at full and quarter-strength concentrations when used in the ax-frill method of stump sprout control of blue gum eucalyptus in the east San Francisco Bay hills (Hamilton et al. 1976).

Although glyphosate is classified as a broad spectrum herbicide, there is considerable evidence to demonstrate relative degrees of sensitivity and tolerance among various plant species. The factor most easily related to plant susceptibility is rate, method and timing of application and the resulting glyphosate exposure. Limited glyphosate uptake was related to glyphosate tolerance of Norway spruce (Lund-Hoie 1976) and Canada thistle (Gottrup et al. 1976).

Fish and Other Aquatic Animals

The following studies have been summarized by Sandquist (1979). The acute toxicity (96-hour TL50) of technical glyphosate to rainbow trout was 38 ppm. The TL50 for bluegills was 78 ppm. Roundup TL50s were 48 and 24 ppm for rainbow trout and bluegills. In carp, the TL50s at 48 and 96 hours were 119 and 115 ppm, respectively.

Bluegills were exposed to a mean concentration of 0.612 mg/liter. On day 1, the mean tissue concentration (to residue measured as carbon 14) was 1.02 mg/kg. No increase in tissue concentration was observed during the subsequent 28-day test period. A bioconcentration factor (edible tissue ^{14}C/water ^{14}C) of 1.6 was calculated.

Short-term tests with water fleas (Daphnia sp.), Atlantic oysters, grass shrimp, and fiddler crabs have been conducted. The 48-hour LC50 of Roundup in Daphnia sp. was 192 ppm (on a volume/volume basis). No effect on embryonic development of oyster larvae was observed following 48-hour exposures to glyphosate concentrations of 10 ppm. The 96-hour TL50s in grass shrimp and fiddler crabs were 381 and 934 ppm, respectively. The no-effect levels in those two animals were 210 and 650 ppm.

Wildlife

Results of toxicity testing in several wildlife species have also been summarized by Sandquist (1979). These results are similar to the findings of toxicity testing in laboratory rodents and dogs (Chemical Toxicology section).

The acute oral toxicity (LC50) of technical glyphosate in an 8-day study in bobwhite quail was greater than 4,640 ppm in diet. When carbon-14 glyphosate was given to quail no storage of ^{14}C was found in muscle and fat. Liver and kidney contained trace levels of radioactivity. An 8-day study in mallard ducklings indicated that the acute dietary LC50 was greater than 4,640 ppm technical glyphosate in diet.

Technical glyphosate and Roundup have a 48-hour LD50 of 100 mg/bee following topical or oral (feeding) administration.

Livestock

No evidence of special hazards to livestock is available. Very rapid clearance of glyphosate in dairy cows has been observed (Monsanto 1980).

Hazard Assessment for Current Use Practice

Glyphosate is an effective herbicide which can be used in vegetation management to accomplish the selective or general elimination of target vegetation. Application rate, method and timing determine susceptibility of plants to glyphosate due to its broad spectrum herbicidal activity.

Roundup is rated as moderately irritating and care should be taken to minimize contact with skin, eyes, and clothing. The human hazard signal word on the herbicide label is therefore WARNING. The Roundup Herbicide Bulletin (Monsanto 1980) reports that evaluation of the irritation potential of use concentrations has been made. The Bulletin states: "Personnel handling and human patch test results indicate that irritation to normal skin from contact to use solutions of Roundup is highly unlikely."

Metabolic studies using laboratory rodents, domestic animals including cattle and chickens, and wildlife have shown that glyphosate does not accumulate in tissues. No evidence of carcinogenic, teratogenic, or mutagenic activity has been obtained in toxicity testing.

The environmental fate of glyphosate has been evaluated under a variety of laboratory and field conditions. Glyphosate has been found to be nonpersistent. Glyphosate is relatively immobile in soil and leaching of phytotoxic amounts from treated areas is unlikely. Soil and water microorganisms are active in the degradation of glyphosate to natural products (i.e., carbon dioxide, water, nitrogen, and phosphate). Due to soil adsorption, the phytotoxic effects of glyphosate are limited to those resulting from direct foliar application or from accidental or unavoidable drift of aerosolized herbicide formulation.

Use of Roundup as indicated on the pesticide label results in a low degree of hazard to personnel, the public and the environment.

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MONURON

Chemical Identification

Monuron is the common name for 3-(p-chlorophenyl)-1-dimethyl-urea. Monuron 80 WP is the trade name (Aceto Agricultural Chemicals Corp.) of a wettable powder containing 80 percent monuron as the active ingredient (EPA Reg. No. 2749-60). This formulation is currently used by BPA; the formulation is noncorrosive to equipment, nonvolatile, and nonflammable.

Previously, the identical (80 percent) formulation was available as Telvar (Du Pont trade name, USDA Reg. No. 352-246). Telvar's registration was terminated recently and Telvar is no longer available. BPA has received special permission from the U.S. Environmental Protection Agency to use up its existing stock of Telvar in accordance with label instructions for substation weed control during Spring 1982.

Monuron is a white crystalline solid which melts at 174°C to 175°C. The specific gravity of monuron is 1.27. The vapor pressure of monuron is 5×10^{-7} mm Hg at 25°C and 3.4×10^{-6} mm Hg at 50°C. The limit of water solubility of monuron is 230 ppm.

Acton in Vegetation

Monuron is an effective general herbicide for the control of many annual and perennial grasses and herbaceous weeds on noncropland areas. Monuron is more effective as a soil application than as a foliar application (McCall 1952).

Monuron is readily absorbed through the root system and is taken up to a limited extent by stems and foliage (Haun and Peterson 1954). When application is made to upper plant parts, little or no monuron is detected below the lowest point of application. Translocation from the roots is primarily upward through the xylem via the transpiration stream (Ashton and Crafts 1973).

The primary toxic action of monuron in plants results from inhibition of photosynthesis (Ashton and Crafts 1973). Within a few days following exposure, plants exhibit leaf-tip dieback followed by progressive chlorosis and reduced growth.

Urea herbicides including monuron are metabolized by higher plants. Species differences in relative rates of metabolism may be related to selective toxicity. Demethylation is the primary detoxication reaction. Hydrolysis of monuron involves a deamination and a decarboxylation which yields p-chloroaniline (Smith and Sheets 1967; Swanson and Swanson 1968). Others (Frear and Swanson 1972; Geissbuhler 1969) failed to demonstrate aniline metabolites and suggested that previous reports of aniline metabolites may have reflected sample impurities or breakdown by photochemical or microbial degradation before absorption by plant tissues. The water soluble metabolites are formed by reaction with natural consitutents and are not bioaccumulated (Kearney and Kaufman 1976).

Utilization by BPA

Monuron is used by BPA at substations to control weeds. BPA used on the average the equivalent of 270 pounds of monuron (active ingredient) per year since 1978, and proposes to use 1,370 pounds in 1983.

An aqueous mixture of monuron is applied with portable sprayers to the soil surface shortly before weed growth begins. Dilution and application provide an effective application rate of monuron (active ingredient) between 1,350 and 3,600 mg/m^2.

Chemical Fate and Distribution in the Environment

Soil

Urea-based herbicides such as monuron are relatively persistent in soils. Principle factors affecting persistence of the ureas in the soil are microorganism decomposition, leaching, adsorption on soil colloids, and photodecomposition. Photodecomposition is an important consideration only with respect to the amounts of herbicide which remain on the soil surface. Volatility and chemical decomposition are of minor importance.

The behavior of monuron in different soil types has been reviewed by Sheets (1964). Adsorption on organic particles and various types of clay colloids controls the removal of monuron from soil and influences the rate of microbial decomposition. Adsorption is lowest in sandy soils and highest in soils with high organic matter content. Other studies have shown that inorganic solutes in soil water reduce the solubility of monuron (Hurle and Freed 1972).

The presence of solutes in soil water will tend to favor adsorption and persistence of urea herbicides in soil. Monuron is more tightly adsorbed to soil than diuron (Kearney and Kaufman 1976).

More strongly adsorbed herbicides are less available as components of the soil solution to biological systems including microorganisms and plants. Monuron is degraded less readily than diuron (Sheets 1958). Since rates of degradation also will be dependent upon the kind and number of microorganisms present, binding via adsorption alone cannot predict persistence. Conditions favoring microbial growth and monuron degradation include elevated temperatures, high moisture content, presence of organic matter, and soil cultivation (Kearney and Kaufman 1976). Rates of disappearance of monuron are slower in sterilized soils than in nonsterilized soils. Hill et al. (1955) have isolated a Pseudomonas species capable of utilizing monuron as sole carbon source. Other common soil bacteria, including Xanthomonas, Sarcina, and Bacillus, can utilize monuron as a carbon source (Hill and McGahen 1955). Survey studies (Borner et al. 1969) have shown that a large number of soil fungi and bacteria can degrade monuron and other urea herbicides.

Water

The persistence and distribution of monuron in an aquatic environment was studied by Frank (1966). The distribution of herbicide between soil and water and the persistence in water and submersed soil were measured. Soil from an irrigation canal was covered with water and stabilized for one month. Wettable powder suspensions were applied (40 pounds/acre). Appreciable losses were evident after 16 weeks. Forty percent of the monuron was lost between the 16th and 32nd weeks. Monuron moved quickly down in the soil, but more monuron was found in water than in soil during the first 32 weeks.

Eichelberger and Lichtenberg (1971) studied monuron persistence in river water during an eight-week test period. The river received domestic and industrial waste and farm runoff. The monuron concentration decreased in closed glass containers from 80 percent at zero time to 40 percent after one week to 20 percent after four weeks. None was detected after eight weeks.

Mikhailov (1967) measured monuron in reservoir waters to establish permissible concentrations. Monuron could be detected by smell at 5 ppb (mg/liter) and by taste at 6.6

ppb. The smell of monuron was stable. Monuron increased the biochemical oxygen demand (BOD) and inhibited ammonification and nitrification at 25 to 50 ppb. Based upon these results the permissible concentration was established at 5 ppb.

Growth of cotton plants was reduced when they were watered with water containing 1.04 and 0.44 ppb monuron. The response was dose-dependent. Bersonova (1967) concluded that irrigation systems being treated with herbicides could be used for irrigation of cotton plants because concentrations used in field practice were below the toxic threshold.

Air

The vapor pressure of monuron is very low, therefore little monuron should volatilize into the atmosphere. A method has been developed for sensitive monitoring of air for monuron (Vengerskaia and Kur 1969), but this procedure has not been utilized to determine amounts of monuron in air associated with vegetation management.

Chemical Toxicology in Animals and Humans

Acute

Monuron has a low order of acute oral toxicity in the rat. The LD50 was determined in two separate studies to be 3,600 mg monuron/kg (Zapp 1955) and 3,400 mg/kg (Hodge et al. 1957). All of the rats that died had pulmonary edema (fluid accumulation) and congestion. Survivors showed signs of methemoglobinemia (presence of a certain hemoglobin incapable of binding oxygen) including cyanosis (blue skin), enlarged dark spleen, and compensatory red blood cell formulation in spleen and bone marrow. Kidney pathology was sometimes present.

Technical monuron (94 percent) in diet was fed to rats (Boyd and Dobos 1969). The LD50 was 1,480 $\pm$ 210 mg monuron/kg when fed in standard laboratory chow. Animals given low protein diets (3.5 percent casein) from weaning to two months of age were 3.5 times more sensitive than rats fed normal (26 percent) casein diets (LD50 950 mg/kg vs 2,880 mg/kg).

Other estimates of the approximate lethal dose (ALD) have been made in rats (ALD 7,500 mg/kg), guinea pigs (ALD 670 mg/kg), rabbits (ALD 1,500 mg/kg) (Zapp 1955).

Subacute studies in rats were also conducted (Zapp 1955). Dosages of 1,500 mg/kg were given five times per week for eight days by stomach tube. Discomfort, weakness, and weight loss were observed, and pulmonary edema and damage to liver, kidneys, and spleen were also reported. Rats given a lower dosage (500 mg/kg) also showed discomfort, weight loss, and cyanosis after two weeks of treatments. During 10 days of post-treatment observation, weight was regained to almost the original level. Spleens were dark, enlarged, and congested.

A 20 percent suspension of monuron in diemethyl phthalate applied to shaved rabbit skin gave an exposure equivalent to 2,250 mg/kg in eight hours. The rabbit showed neither clinical signs of toxicity nor clinical pathology when killed 11 days later (Zapp 1955). A 33 percent water paste was practically nonirritating and did not cause allergic skin sensitization following application to the skin of guinea pigs. It was concluded that moderate skin contact would not be harmful (Zapp 1955).

In one method of interpreting toxicity data, it is presumed that acute (LD50) toxic doses for rat populations are similar to that for human populations. Given an LD50 of 3,500 mg/kg for rat populations, it is presumed that the LD50 dose of monuron (active ingredient) for a population of 165-pound humans would be 9.3 ounces (0.056 oz/lb).

Chronic Effects

Six-week feeding trials were conducted using diets containing 0.005, 0.05, and 0.5 percent (50, 500, and 5,000 ppm) monuron (Zapp 1955). At the lowest level, no differences between treated and control groups were observed with respect to food consumption, weight gain, and clinical chemistry. At necropsy, two of five males showed evidence of methemoglobinemia and/or compensatory red blood cell formation. No significant pathology was observed in females. At the intermediate level, the only toxic effects noted were methenoglobinemia and compensatory red blood cell formation in the spleen of both males and females. At the highest level, food consumption and weight gain were reduced. Signs of toxicity were similar to those observed in acute studies although no mortality occurred.

Subsequent two-year feeding studies utilized diets containing 0, 25, 250, and 2,500 ppm monuron (Hodge et al. 1958). No effects were observed at 25 and 250 ppm. At the highest level, weight gain was reduced. Survival time was not affected by the treatments although 70 to 90 percent of the

groups had died by the end of the second year due to upper respiratory infection. At necropsy, liver and spleen weights were higher in treated than control rats. No histopathological effects were observed in either kidney, heart, brain, lung, spleen, liver, adrenal, bladder, gonad, stomach, intestine, or bone marrow. The incidence of tumors was no higher in treated than in control groups.

Feeding studies using two beagle dogs revealed no effects. Groups of a male and a female were given 0, 2.5, 12.5, and 25.0 mg monuron/kg/day for one year (Hodge et al. 1957).

Reproductive studies have been conducted in rats (Sherman and Culik 1971). Males and females were fed either 0, 125, or 2,500 ppm monuron in the diet for three generations. Two litters were produced per generation. Average litter size was lower in the 2,500 ppm group than in control and 125 ppm groups. Male and female rats from the third generation were evaluated and no treatment-related histopathology was observed.

Monuron was included in a teratogenicity survey of pesticides (Mrak 1969). No significant increase in fetal anomalies was seen in three strains of mice given 215 mg/kg.

Two carcinogenicity studies (Rubenchik 1970; Innes et al. 1969) have been described in addition to the earlier observations of Hodge et al. (1957). Innes et al. (1969) used maximum tolerated doses in two hybrid mice strains of monuron as one of 130 chemicals tested for carcinogenicity. Males and females were given 215 mg/kg/day by stomach tube from day 7 until weaning at four weeks of age. The same daily dosage was administered in the diet until necropsy at 18 months of age.

Mice fed monuron had elevated tumor incidence, but not significant enough to place the compound in the carcinogenic category. Further evaluation was recommended. Rubenchik et al. (1970) administered monuron to rats and two strains of mice. Increased tumor incidence was observed.

A study of the chronic toxicity of monuron under the sponsorship of the National Cancer Institute is in progress using rats and mice at maximum tolerated and half-maximum tolerated dosages (EPA 1975). A review by the International Agency for Research on Cancer (1976) concluded that monuron was carcinogenic.

Potential Impact on Nontarget Organisms

Vegetation

Increased incidence of mildew Erysiphe graminis in winter and spring wheat after application was reported (Brandes and Heitefuss 1971). The response was related to the stage of plant development, light intensity, and the amount of herbicide treatment. The authors concluded that monuron caused increased disease susceptibility by changing the physiological condition of the host plant.

Fish and Aquatic Organisms

Acute toxicity data include LC50s or median tolerance limits (24- or 48-hour) which range from 10 to 180 ppm in six species (EPA 1975). The 48-hour LC50 of monuron in rainbow trout was 100 ppm and the 48-hour TLm in coho salmon was 110 ppm. The trichloroacetic acid formulations of monuron are more potent than wettable powders to bluegills, brown bullheads, sunfish, and largemouth bass (Walker 1965). Goldfish survived five-day exposures to 59.5 ppm monuron and 35-day exposures to 14.9 ppm (Lawrence 1962). Edson (1958) estimated the three-hour approximate safe upper limit of monuron to be 20 ppm.

Komarovsky and Popovich (1971) observed 46 percent mortality and decreased hemoglobin and hematocrits in fish exposed to monuron (initial concentration 20 ppm) for 75 days.

Fish mortality (not quantified) was observed in small golden shiners exposed in the field to 20 ppm monuron. Bond et al. (1959) reported no mortality in either coho salmon, frogs, or tadpoles exposed to 5 and 10 ppm monuron.

In adult white shrimp, no death, paralysis, or loss of equilibrium were observed at the highest concentration tested (48 hours; 1 ppm; Butler 1963). Two ppm monuron reduced rate of shell growth (12 percent) in the eastern oyster after an exposure of 96 hours. Monuron had 48-hour and 12-day TLms of greater than 5 ppm in the eastern oyster and hard-shelled clam (Davis and Hidu 1969).

Concentrations up to 20 ppm monuron did not cause mortality in amphipods or isopods (Springer 1957). Immobilization of water fleas (Daphnia magna) occurred at 106 ppm in water containing 1 ppm non-ionic surfactant (Tween 20) (Crosby and Tucker 1966). Nishiuchi (1974) reported a three-hour TLm for D. pulex of greater than 0.05 ppm.

Walker (1965) evaluated monuron and other urea herbicides as aquatic herbicides. Monuron was less toxic than other representatives of this group. Walker (1965) concluded that the herbicides "...did not appear to seriously reduce fish-food organisms, and no fish mortalities were observed under field conditions at concentrations up to 10 ppm of monuron".

Wildlife

Dietary LC50s of monuron were determined in four species of birds. Two- to three-week-old birds were fed treated diet for five days followed by a three-day observation period. The LC50 in ring-necked pheasants was 4,682 ppm. The LC50 was greater than 5,000 ppm in bobwhite quail, Japanese quail, and Mallard ducks (Heath et al. 1972).

Atkins et al. (1973) reported that monuron was moderately toxic to honeybees. The LD50 of monuron was 110 micrograms per bee when the herbicide was applied as a dust.

Livestock

Cattle and sheep were dosed by either drench or capsule and chickens by capsule only. Signs of poisoning were weight loss and diarrhea in cattle and sheep at high daily dosages (500 mg/kg and 100 mg/kg, respectively). At necropsy, the lungs of the sheep were engorged with blood and respiratory mucosa were congested. Liver and kidneys were congested and enlarged. Meningeal vessels of the brain were engorged. Chickens at 10 mg/kg/day for 10 days generally showed congestion of the intestinal mucosa and atrophied spleens.

Feed contamination by monuron could be toxic to livestock. Rates of application in excess of three pounds/acre would be hazardous to chickens. Very high application rates (80 pounds/acre) would be hazardous to cattle and sheep (Palmer and Radeleff 1969).

Hazard Assessment for Current Use Practice

Herbicides containing monuron are used to a limited extent by BPA for the control of weeds at substations.

Based upon testing in laboratory animals, monuron has been shown to have a low order of toxicity. Testing related to acute oral (ingestion), skin, and eye exposures is especially important to assess the hazard to persons who must

work with concentrated formulations. As a result of this testing, the Human Hazard Signal Word CAUTION is displayed on the herbicide label.

Metabolic studies using soil microorganisms, plants, laboratory rodents, and domestic animals have shown that monuron is extensively metabolized and is not retained in tissues. No evidence of teratogenic or mutagenic activity has been reports.

Monuron has caused excess tumors in experimental studies in rodents and is therefore classed as an animal carcinogen. The carcinogenic potential of monuron is currently under evaluation by the Environmental Protection Agency.

No epidemiological studies with workers have been performed to assess the extent of human exposure associated with the use of monuron. As is always the case, exposure can be kept at a low level by careful adherence to application instructions. The extremely small scale of monuron useage by BPA minimizes any real or apparent threat to health.

The environmental fate of monuron has been studied under a variety of laboratory and field conditions. Microbial degradation is the primary factor responsible for the reduction of monuron concentrations in soils. The extent of soil adsorption increases as clay and/or organic matter increase. Leaching is not an important route of loss except in sandy soils. Losses by volatilization and photodecomposition are negligible. Monuron is sufficiently persistent that phytotoxicity will be retained at least one season, and longer at higher rates of application.

Use of monuron as indicated on the pesticide label and in limited quantities at substations results in a low degree of hazard to personnel, the public, and the environment.

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PICLORAM

Picloram is the common name of 4-amino-3,5,7-trichloropicolinic acid. Tordon is the name of the picloram-containing herbicide that is marketed by the Dow Chemical Company, Midland, Michigan. Tordon 10K pellets (EPA Reg. No. 464-320AA-264) is formulated for soil application for control of undesirable woody plants. The herbicide contains 11.6 percent picloram as the potassium salt. Tordon 101 (EPA Reg. No. 464-306AA-264) is a mixture containing picloram and 2,4-D. The triisopropanolamine salts of picloram and 2,4-D constitute 10.2 and 39.6 percent of the herbicide. A separate background statement has been prepared for 2,4-D.

Picloram is a white powder with a chlorine-like odor at room temperature. Chemical decomposition occurs before melting (at approximately 215°C). The vapor pressure of picloram is 6.16×10^{-7} mm Hg at 35°C and 1.07×10^{-6} mm Hg at 45°C. The solubility of picloram (grams per 100 ml solvent) in acetone, acetonitrile, benzene, and methylene chlorine is 1.98, 0.16, 0.02, and 0.06, respectively. The limit of water solubility of picloram is 0.043 g/100 ml (or 430 ppm).

Action in Vegetation

As a potassium salt (Tordon 10K) (EPA Reg. No. 464-320AA-264), picloram is effective against shrubs, hardwoods, and conifers. When mixed with 2,4-D and formulated as Tordon 101 (EPA Reg. No. 464-306AA-264), the herbicide is effective against annual and perennial broadleaf weeds and woody plants (shrubs, hardwoods, and conifers).

Picloram is readily absorbed by both foliage and roots. It is translocated in the phloem and xylem. Signs of phytotoxicity are prominent in leaves, which develop irregularly. The tips of new leaves may develop into extensions of the midrib and thickening of the mesophyll. Leaves may become puckered, cupped, or stunted. Tissue growth along the stem may occur. Cracking or splitting of the stems and deterioration of roots may be observed before the plant dies (Ashton and Crafts 1973). Picloram is classed as a auxin-like, growth-inhibiting herbicide.

Picloram is remarkably stable in plants. Sharma and Vanden Born (1971) found less than 1 percent of the total dose

applied to Canada thistle, soybean, and corn to be degraded to carbon dioxide in 20 days.

Utilization by BPA

Picloram (Tordon) is used by BPA on rights-of-way and at substations in noncrop areas for the management of woody species (shrubs and trees). BPA used on the average the equivalent of 3,426 pounds of picloram (active ingredient) per year since 1978. None is proposed for use in 1983.

Tordon 10K is used on rights-of-way and at substations where woody plants are a nuisance. It is applied by portable spreaders onto the soil surface beneath the target vegetation at any time the soil is not frozen. Effective application rate of picloram (active ingredient) is between 260 and 1,080 mg/m^2.

Tordon 101 is used to control weeds and woody plants on rights-of-way and at substations. The herbicide is applied as a high-volume, ground-based broadcast spray, as a directed (spot) spray, as an aerial treatment broadcast by a helicopter, or with single-stem and cut-stump treatment devices. Application is made after foliage is well developed, but while weeds and brush are actively growing. Dilution (with water) and broadcast application rates provide an effective application rate of picloram (active ingredient) between 56 and 168 mg/m^2. When applied directly to single stems or cut stumps, dilution yields 60 and 240 grams/liter picloram and 2,4-D, respectively.

Chemical Fate and Distribution in the Environment

Soil

Losses of picloram from soil were measured by Goring et al. (1965). Losses ranging from 58-96 percent occurred within one year and 78 to 100 percent within two years. At two locations, highest concentrations were found in the upper 30.5 cm of soil and detectable amounts were recovered at soil depths of 121.9 cm below the soil surface. Similar observations have been made by Keys and Friesen (1968) and Scifres et al. (1969).

If rainfall occurs shortly after picloram application, significant amounts may be removed from treated areas by surface runoff. In one case, picloram was applied at a rate of 1.12 kg/ha. Heavy rainfall two days later resulted in

concentrations of 26.2 to 89.7 ppb picloram in runoff water. Eight months after treatment, runoff water contained less than 1 ppb.

Picloram has low vapor pressure and is not lost to a significant extent from soil by volatilization (Youngson et al. 1967).

Picloram can be easily leached from soil by heavy rainfall and is very persistent during its downward movement into the soil column (Hamaker et al. 1963, cited in Foy 1976).

Minimal soil adsorption of picloram occurs under neutral or alkaline conditions in sandy loam soil. Adsorption increases with decreasing pH, increasing organic matter, and increasing levels of hydrated iron and aluminum oxides (Hamaker et al. 1964, cited by Foy 1976).

Nonbiological chemical breakdown of picloram occurred in soil at high soil:herbicide ratios (10:1) (Hance 1969).

Sterilization of soil eliminated picloram degradation in soil. Considerable variability in the extent of degradation was found in a series of natural soils and in pure cultures of a variety of soil organisms. Increasing temperature and organic matter increased the rate of picloram decomposition. The amounts degraded were very small indicating that the herbicide was not utilized as an energy source. After one year, 80 to 97.5 percent of radioactivity was present as the parent compound (Youngson et al. 1967).

The persistence of picloram in northwest soils has been studied by Norris (1971, 1972, 1973, 1974, 1975, 1976, and 1979). These studies were conducted to evaluate the fate of selected herbicides used in BPA vegetation management programs. Chemicals studied in addition to picloram included 2,4-D and dicamba. Test sites were chosen to represent typical settings where chemical vegetation management was practiced. The use of picloram resulted in detectable soil residues in litter and upper soil layers but it was not found below the 18-inch level of the soil profile. The residues were detectable for periods up to three years.

At Reston-Fairview, Carson Tap, and Longview-Astoria, soil samples were collected five or six months after Tordon 101 application at rates of two gallons/acre. Herbicide residues were usually found in surface litter or the 0- to 6-inch soil layer. In one case, residues of picloram were detected in the 6- to 12-inch layer. The amount present in soil at lower depths was less than the amount at the surface. The two lowest layers contained no picloram residues. When these three sites were sampled about a year

later (16 to 18 months postspray), the residue levels, in nearly all cases, were similar to those measured at six months postspray (Norris 1972). Some residue levels at Reston-Fairview apparently were higher in the upper litter layer in the second set of soil tests. No evidence of leaching was obtained. A third set of long-term samples was obtained 24 months postspray at Reston-Fairview and 25 months postspray at Longview-Astoria (Norris 1973). (The Carson Tap site was resprayed in 1972 and was, therefore, not sampled in 1972.) Two of three litter samples at Reston-Fairview contained detectable (0.02 and 0.05 ppm) picloram. None was present at Longview-Astoria. Analysis of soil to depths of 30 inches provided no evidence of leaching (not surprising since levels in upper soil levels were very low). Only the Reston-Fairview site was sampled in May 1973 (three years postspray). At that time, picloram residues were no longer detectable in litter or soil (Norris 1974). The results obtained at these sites are quantitatively and qualitatively similar to those obtained at long-term study sites that were established later by Norris (1972, 1973, 1974, 1975, 1976, 1979).

The persistence of picloram in Oregon and Washington soils that had been sprayed at similar rates (about two gallons/acre) was also established. Picloram was not detected 31 and 43 months postspray at two sites in Oregon, but it was detected in upper soil layers (to 12 inches) 52 months postspray at Reedsport-Fairview and seven months postspray at Florence Top tower. At the latter site, herbicide was applied three times between 1965 and 1970. The levels detected (to 0.15 ppm) gave no indication of herbicide build-up in the soil. Analysis of soil from four Washington sites treated 22 to 58 months earlier did not reveal significant residues.

Water

Picloram has sufficient mobility in the environment that the early runoff waters from treated areas have frequently been shown to contain detectable residues.

First fall runoff waters contained up to 78 ppb picloram following summer treatment of 67 percent of an Oregon watershed. No residues were detected in runoff from areas less extensively treated. After late October, no picloram was detected (Norris 1971). In California chaparral, an August application of one, two, and four pounds/acre picloram resulted in first runoff water concentrations of 0.1, 0.5, and greater than 0.5 ppm picloram, respectively. After 15 inches of rainfall, picloram concentrations were reduced to 0.01, 0.03, and 0.03 ppm (Green 1970).

Water that collected in ponds adjacent to treated areas contained up to 184 ppb picloram when rainfall occurred within two weeks of application (Haas et al. 1971). When the first rain was six weeks after the application, the maximum pond concentration was 28 ppb. During the first 100 days, the concentration decreased rapidly and stabilized at approximately 5 ppb.

Following treatment of an 80-acre area with one pound/acre, first runoff water contained 29 ppb picloram. After five months, detectable levels were present in water 0 and 0.5 miles downstream from the site. No picloram was detectable during a two-year observation period in well water from adjacent areas (Haas et al. 1971).

The outflow of a 16-acre Oregon watershed contained 0.28 percent of the total picloram during the three years following the application of picloram at two pounds/acre. The first water to flow from the watershed contained 20 ppb picloram. No herbicide was detected after seven months. Picloram was also applied to a dry stream channel which represented 0.21 percent of the area of the watershed. Norris et al. (1976) concluded that picloram in the outflow water resulted primarily from material applied to dry stream channels.

Herbicide monitoring studies conducted by BPA have revealed little or no contamination of streams which cross treated rights-of-way (Norris 1979 and previous reports). Water in two of the study sites established in 1970 was sampled approximately six months and 16 or 24 months postspray. The sites were Carson Tap and Longview-Astoria. No residues of either 2,4-D or picloram were found in water at either of the two sites.

In 1971, four additional long-term sites were selected and the sampling program was extended by establishment of spray interception discs to obtain measurement of chemical input. The sites designated Monroe-Custer and Snohomish-Bellingham were sprayed with two gallons Tordon 101/acre. Water collected from the Snohomish-Bellingham site seven months after the spraying contained no detectable 2,4-D and a trace of picloram.

Water from the Coos Tap Tower site was sampled in a more extensive program. No evidence of 2,4-D or picloram in water was obtained, indicating that neither leaching nor surface runoff of picloram was occurring. At the fourth site, Vantage-Raver, water was sampled from a spring, a U.S. Bureau of Reclamation canal, a creek crossing the right-of-way, and from a domestic well. The spring sample (one

only) contained 0.003 ppm picloram immediately after the helicopter spray operation. 2,4-D was present in three creek samples and in the spring water (0.020 ppm). No picloram residues were detectable in any of the other water samples taken at the Vantage-Raver site. Similarly, no water residues were detected at the Big Cliff-Detroit study site two and four months after Tordon application.

These monitoring studies showed that trace amounts of herbicide can enter water as a consequence of spray operations. The amounts detected do not constitute biologically significant exposures (see Potential Impact on Nontarget Organisms).

Buffer strips of 100 feet have been shown to be an effective means to minimize herbicide entry into streams. At the Olympia-Port Angeles site, unprotected creek water contained 0.010 ppm 2,4-D and 0.007 ppm picloram immediately after the spray operation. In the area in which the buffer zone was utilized, no herbicide was detectable in creek water. After 24 hours, picloram was detectable in water at both sites, but 2,4-D was present only in water from the unprotected creek. The amounts detected (0.007 ppm immediately after spraying to 0.003 ppm in 24 hours) were not of biological significance to aquatic organisms or to downstream water users (Norris 1973).

A more intensive sampling protocol also has been used by Norris (1976). The John Day-Marion site was treated in July 1976 with 2,4-D amine (six pounds) and picloram (one pound) and water was sampled for nearly five months. Herbicide was present after 30 minutes in low concentrations (0.044 ppm 2,4-D and 0.015 ppm picloram). After 90 minutes, 2,4-D levels dropped to 0.003 ppm and no picloram was detectable (less than 0.003 ppm). The other samples of the series also did not contain detectable herbicide. The residues likely resulted from the spray operation itself. No evidence of long-term movement of the herbicide from treated soil and vegetation was obtained.

On July 5, 1972, a contractor's truck overturned on a right-of-way access road near Olympia, Washington, spilling 1,250 gallons of 10% Tordon 101 in Norbak carrier. Although the accident occurred on a steep slope (60-90% slope), 80% of te spilled material remained as a solid, gelatinous mass in the first 50 feet of spill. Following clean-up, an estimated 25 gallons of Tordon 101 remained on the spill site, and resulted in low-level contamination of the well water of a residence near the spill site on the Olympia-Port Angeles line (Norris 1979). In November 1974, the amount of picloram in unfiltered well water was 0.16 ppm and in April 1978 a similar sample contained 0.0016 ppm. These data

indicated that picloram was a persistent contaminant in the water supply as a result of the accident. Charcoal filtration was used to reduce the amount in water in one case from 0.000334 ppm (.334 ppb) to 0.000042 ppm (.042 ppb) (letter of August 1, 1978 - Norris to Grassel).

Air

Volatilization is of minor importance in the reduction of soil levels of picloram. Some picloram may be degraded during aerial application by photodecomposition. Studies of photodecomposition on soil surfaces and in water have amply demonstrated that sunlight can supply sufficient energy to degrade picloram (Newton and Dost 1981). The quantitative significance of these transformations is neither known nor has it been authoritatively estimated.

Chemical Toxicology in Animals and Humans

Acute Effects

Picloram has a low order of acute oral toxicity in animals. The acute oral LD50 in the rat is 8,200 mg/kg. In mice, the LD50 is within the range of 2,000 to 4,000 mg/kg. The approximate LD50s in rabbits, guinea pigs, and chicks are 2,000, 3,000, and 6,000 mg/kg. The LD50s of picloram are greater than 1,000 mg/kg in sheep and greater than 750 mg/kg in cattle (WSSA 1979).

Inhalation of picloram dusts may be irritating but is not likely to cause illness (WSSA 1979).

The LD50 for rabbits resulting from skin application is greater than 4,000 mg/kg (the highest level tested). Mild skin irritation, but no skin sensitization, has been observed in humans. Skin absorption is "not likely" (WSSA 1979).

Moderate eye irritation which heals readily is listed as a hazard in WSSA (1979). No corneal injury is likely (WSSA 1979).

Nausea may result from ingestion of massive amounts of picloram (WSSA 1979).

In one method of interpreting toxicity data, it is presumed that acute (LD50) toxic doses for rat populations are similar to that for human populations. Given an LD50 of 8,200 mg/kg for rat populations, it is presumed that the

LD50 dose of picloram (active ingredient) for a population of 165-pound humans would be 21.7 ounces (0.131 ox/lb).

Chronic Effects

Ninety-day studies in rats using diets containing up to 1,000 ppm produced no ill effects (McCollister and Leng 1969). Two-year feeding studies in rats and beagle dogs resulted in daily exposures of 15 to 150 mg/kg. No adverse effects were detected with respect to weight gain, food consumption, behavior, mortality, hematology, and clinical chemistry (blood and urine).

On the basis of the above study, Dost (1983) considered 80 mg/kg/day to be the no-effect reproductive level for picloram in a worst-case analysis of environmental exposure.

Over 90 percent of a dose of ^{14}C-carboxy picloram (97 ppm in diet) was eliminated within 48 hours of the feeding. Tissues did not accumulate the herbicide and decarboxylation was not an important degradation process (McCollister and Leng 1969). McCollister and Leng (1969) suggested that rapid clearance and elimination in urine minimized picloram metabolism in liver.

The reproduction toxicity of picloram was studied (Thompson et al. 1972) in rats given 500, 750, and 1,000 mg/kg on days 6-15 of gestation. Twenty-five rats were killed and studied for fetal abnormalities, and ten were allowed to deliver pups which were studied for postnatal effects. Ten maternal deaths occurred at the higher dosages, and in the group held for postnatal observation for maternal deaths at the higher dosages. Evidence of delayed development of the fetuses but no teratogenic activity was associated with the higher dosages. No postnatal effects were observed (Thompson et al. 1972).

An earlier three-generation study was conducted using a lower dose and longer exposure period. Four male and 12 female rats were given 150 mg picloram/kg/day beginning 28 days before the first mating. No effect on reproductive capacity was detected (McCollister and Leng 1969). Some stillbirths in the first generation and reduced fertility in the second generation were observed but their incidence was not related to dose.

In a mutagenicity screen of 110 herbicides, Anderson et al. (1972) found that picloram did not produce point mutations in eight histidine-requiring strains.

Carcinogenicity studies have been performed as part of long-term feeding experiments by the manufacturer and later

as elements of work of the National Cancer Institute. The results of two carcinogenicity studies failing to demonstrate increased tumor incidence have recently been declared invalid by EPA, and a third test has been found to have serious deficiencies. EPA has ordered the tests to be repeated. In an unrelated study, no evidence of increased tumor incidence was obtained at doses of 150 mg/kg/day fed to rats (NRC Canada 1974).

Fifty mice and 50 rats of each sex were used at time-weighted average doses of 5,062 and 2,531 ppm in the mouse diets and 14,875 and 7,437 ppm in the rat diets. The rats and mice were exposed to the procedure for 80 weeks. They were necropsied after an additional 33 and 10 weeks, respectively. An increase in thyroid adenomas (benign glandular tumor) in rats was not statistically significant, and the effect was not observed in mice. A significant increase in neoplastic (tumorous) nodules was observed in female rats. It was concluded that picloram was not carcinogenic in mice or in male rats. High sustained doses of picloram could induce benign tumors in female rats (Innes et al. 1969).

Potential Impact on Nontarget Organisms

Vegetation

Picloram is a potent herbicide for general woody plant control and control of most annual and perennial broadleaf weeds. Most grasses are resistant, making it feasible to control broadleaf weeds in grass crops. Broadleaf crops are sensitive except Cruciferae (e.g., cabbage, brussel sprouts, and broccoli).

Foliage and bark samples from Bonneville-The Dalles line (between structures 3/5 and 3/7) were collected from damaged trees off the right-of-way. The foliage of damaged trees contained picloram (up to 6.9 ppm). The degree of damage was related to the extent of herbicide contamination. The ultimate source of picloram was not established although picloram had been used on the right-of-way. It was speculated that either tree-to-tree transfer via root grafts or runoff may have been responsible for the damage (Norris 1975).

Douglas-fir seedlings were damaged by residual picloram in soil resulting from Tordon 101 spraying (1.5 gallons/acre). The seedlings were planted about 18 months after the spraying and foliage was sampled a month later. Soil contained 0.08 (under heavy litter) and 0.33 ppm (bare soil) picloram and foliage contained 0.11 to 0.17 ppm. Damage was not

severe on most trees and the distribution of damaged trees was irregular (Norris 1972).

Fish and Aquatic Organisms

Picloram has a low order of acute toxicity to fish and other aquatic organisms. LC50s for picloram acid and its important salts (including potassium and triisopropanolamine) have been measured in a variety of fishes. The 24-hour LC50s of the potassium salt in harlequin fish, channel catfish, and bluegill were 66, 41, and 69 ppm, respectively. The 24-hour LC50 of the triisopropanolamine salt was 297 ppm in rainbow trout (Foy 1976).

Some aquatic invertebrates have been tested for sensitivity to picloram. Stonefly nymphs were killed at high concentrations with an LC50 of 120 ppm. The 48-hour LC50 in amphipods was 48,000 ppm (well above the solubility limit). Daphnia were not affected in 24-hour tests at 380 ppm but 95 percent were killed at 530 ppm (Foy 1976). Eastern oysters exposed to 1 ppm picloram for 48 hours did not show any adverse effects on shell growth (Pimentel 1971).

The animals in an aquatic microcosm consisting of algae, Daphnia, guppies, and goldfish were not affected by a ten-week exposure to 1 ppm picloram (Foy 1976).

Woodward (1976) has shown that low concentrations of picloram can reduce survival and growth of lake trout fry. Continual exposure to concentrations of 0.035 ppm began 10 days before hatching and continued for 60 days after hatching. The results indicate that the toxic threshold for early life stages is considerably less than acute toxicity thresholds for mature fish. Continual exposure to concentrations as high as 0.035 ppm is unlikely due to the limited movement of picloram from soil to water.

Subsequent testing on cutthroat trout fry reported by Woodward (1979) found a substantially higher no-effect level; no changes in fry survival or growth were detected at simulated field exposures beginning at 0.29 ppm and decreasing to 0.048 ppm over a 60-day period. Woodward (1979) concluded that picloram should be used in a manner that ensures stream residues do not exceed 0.29 ppm during the first subsequent rainfall after application.

Wildlife

Consistent with its low order of mammalian toxicity, picloram exhibits low toxicity to birds (WSSA 1979).

Japanese quail and bobwhite quail were fed diets containing from 100 to 1,000 ppm but insufficient kill was obtained to establish an LC50. The LD50 was greater than 2,000 mg/kg in young mallard ducks and young pheasants when picloram was administered in capsules. The dietary LD50s were greater than 5,000 ppm to ducks and pheasants (Pimentel 1971).

Livestock and Poultry

Milk of cows contained 0.05 to 2.0 ppm picloram after they had been fed dietary levels of up to 1,000 ppm picloram (18 mg/kg/day) for two weeks. The residue levels dropped to less than 0.02 ppm within two to three days of the cessation of the feeding. Tissue of steers fed up to 1,600 ppm reached a maximum blood level (0.1 to 2.0 ppm) after three days of feeding. Corresponding levels were 0.05 to 5.0 ppm in muscle and fat, 2 to 18 ppm in kidney, and 0.5 to 2.0 ppm in liver. Three days after picloram withdrawal, the kidney level was 0.1 ppm and all others were 0.05 ppm or less. Results of limited dose-response studies indicated that dietary levels of 200 to 400 ppm were required to produce residues of 0.05 to 1.0 ppm in edible tissue (Kutchinski 1969).

The reproductive success of hens and cockerels was not affected by spraying eggs with picloram (Somers et al. 1978).

Recent Developments in Toxicity Testing

Considerable public controversy concerning the use of picloram was prompted by a 1981 paper, "Carcinogenicity of Picloram" (Reuber 1981). In the subsequent issue of the Journal of Toxicology and Environmental Health, institutions with which Doctor Reuber had been associated disclaimed any relationship to the controversial manuscript (Anonymous 1981). These events were related to articles in the public press which focused much public concern on the safety of picloram herbicide.

The primary areas of public concern were the following (USEPA 1982): 1) picloram is one of a small number of pesticides restricted for use by certified applicators; 2) some of the testing data supporting picloram's EPA registration have been found invalid; and 3) one study of rats fed picloram has been interpreted by several scientists as evidence of cancer. In May 1982, EPA officially refuted these allegations (USEPA 1982). General considerations related to the properties and toxicity of picloram were noted. EPA has emphasized that the restricted use classification was assigned due to picloram's high potency in

killing vegetation. Picloram is highly mobile in soil and therefore can travel to the roots of nontarget plants and damage them. The restricted-use classification requiring certified applicators is to minimize damage to nontarget vegetation by runoff or leaching; it is not due to known or suspected health risks.

The other allegations related to picloram use were related to toxicity testing. Registration of a pesticide product requires that the applicant demonstrate that the proposed use will not pose risks of unreasonable adverse effects on human health or the environment. Data on short-term toxicity testing support the current registration. Some studies on long-term (chronic) effects conducted by Industrial Bio-Test (IBT) laboratories were found invalid due to improper laboratory practices. The EPA emphasized that an invalid study does not mean that the chemical is posing risks, but that the results cannot be used to evaluate risk (USEPA 1982).

Two long-term studies by the National Cancer Institute (NCI) were not involved in the IBT problem. One NCI study in mice was considered negative for cancer effects. The other study in rats was considered of questionable value due to laboratory procedures. Benign tumors found in the second study were considered by some scientists as evidence of cancer risk. NCI, EPA, and an independent research firm who reviewed the study did "not regard it (the second study) as providing an answer to the cancer risk question." Further, EPA noted, "even if this study were accepted as positive evidence for potential for inducing tumors, given the high doses needed to produce the effect and the very low potential for human exposure from current uses of picloram, existing uses of the product would not pose a significant risk of increased cancer in the population" (USEPA 1982).

Finally, the EPA press release provided the following summary (USEDA 1982):

> "In sum, the data on short term effects, environmental effects and genetic mutation, as well as one NCI cancer study, support the current registration of picloram. The registrant is conducting a new rat feeding study to clarify the ambiguous results of the second NCI study. The pesticide law places the burden for testing chemicals on their manufacturer, and it is the usual practice for industry to undertake the development of the basic data needed by EPA to make regulatory decisions. We have no current evidence that picloram is posing risks of unreasonable adverse effects to human health or the environment, although more data is needed on long term effects to support this conclusion."

Hazard Assessment of Current Use Practices

Picloram herbicides are used as a directed or broadcast application applied with ground-based and helicopter equipment. Spray adjuvants which thicken the herbicide formulation, e.g., Norbak, are used with aerial applications. Picloram is effective for the control of annual and perennial weeds, brush, and trees in its various formulations. Tordon 101 and Amdon 101 are important products which contain picloram and 2,4-D.

Based upon testing in laboratory animals and in accordance with the Human Health Signal Word System, picloram herbicides are labelled CAUTION since picloram has a low order of acute toxicity.

Chronic toxicity testing using rats and dogs did not reveal harmful effects in two-year studies using dosages of 15 to 150 mg/kg. Teratogenic and reproductive studies using high dietary concentrations (3,000 ppm) were similarly negative (McCollister and Leng 1969). Very limited reproductive toxicity was reported in a later study (Thompson et al. 1972).

When herbicides were screened for mutagenic activity (Anderson et al. 1972), picloram did not produce point mutations. Newton and Dost (1981) summarized studies which indicate that picloram is not carcinogenic in rats and mice. These observations, coupled with the fact that picloram is very rapidly absorbed and excreted by animals and humans, indicate that picloram has very low chronic toxicity hazard. Because of inadequate laboratory procedures in some of the earlier studies, EPA has ordered new carcinogenicity studies.

Picloram is relatively persistent in soils since phytotoxic levels can be present a year or more following application. High organic matter, moisture, and warm temperatures reduce the duration of action of picloram since these conditions favor soil microorganisms which slowly degrade the herbicide. Photodecomposition on surfaces receiving intense sunlight also reduces soil levels of picloram. Losses due to volatility are negligible.

Surface runoff and leaching are both means by which picloram can move from treated soil to water. Well-drained (sandy), light-textured soils and low organic matter favor leaching. Rainfall shortly after application can remove up to five percent of the herbicide from a watershed. In water, dilution and photodegradation are responsible for the rapid reduction of picloram concentrations. The amounts present

in water at any time are not toxicologically significant due to the low toxicity of picloram to aquatic organisms. The herbicide is rapidly excreted and does not bioaccumulate.

Nontarget vegetation can be poisoned with picloram due to its potency and high soil mobility. Toxic amounts of picloram also can be transferred from stem injected trees to adjacent vegetation due to root grafting or exudation (Newton and Dost 1981). Detectable contamination of groundwater has also resulted from accidents (Norris 1979) and agricultural applications (Frank et al. 1979; Ghassemi et al. 1981). These observations illustrate the need to carefully control the release of picloram in the environment.

Based on available information, use of picloram as indicated on the pesticide label and in accordance with guidelines detailed in the Transmission Line Maintenance Standard results in a low degree of hazard to personnel, the public, and the environment.

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PROMETONE

Chemical Identification

Prometon(e) is the common name for 2,4-bis (isopropylamino)-6-methoxy-s-triazine which is the active ingredient of Pramitol 25E and an active component of Pramitol 5PS, both marketed by the Agricultural Division, Ciba-Geigy Corporation (Greensboro, North Carolina). Pramitol 25E (EPA Reg. No. 100-443 AA) contains 25 percent prometone. Pramitol 5PS (EPA Reg. No. 100-479) is a nonselective herbicide that is formulated as a pellet. Pramitol 5PS contains 95 percent active ingredients including prometone (5.00 percent), simazine (0.75 percent), sodium chlorate (40.00 percent), and sodium metaborate (50.00 percent). The latter are subjects of other Background Statements (page A-123 and page A-131, respectively).

Prometone is a white crystalline solid which melts at 91°C to 92°C. The vapor pressure of prometone at 20°C is 2.3×10^{-6} mm Hg and 7.6×10^{-5} mm Hg at 50°C. Prometone is subject to decomposition by ultraviolet light (WSSA 1979). The solubility of prometone at 20°C (parts per million by weight) in acetone, benzene, methanol, and water is greater than 500,000, greater than 250,000, greater than 500,000, and 750, respectively.

Action in Vegetation

Prometone is registered as a nonselective preemergence and postemergence herbicide which controls most annual and perennial broadleaf and grassy weeds in noncroplands.

Rapid absorption of prometone occurs through the roots, and the herbicide is readily translocated throughout the plant in the transpiration stream.

Prometone, like other s-triazine herbicides, inhibits plant growth as a consequence of its inhibition of photosynthesis (Ashton and Crafts 1973). Foliar chlorosis precedes the death of the plant. Sublethal amounts of prometone may stimulate plant growth and increase chlorophyll content.

Utilitzation by BPA

Prometone (Pramitol) is used by BPA to control weeds at substations. BPA used on the average the equivalent of

4,435 pounds of prometone (active ingredient) per year since 1978, and proposes to use 3,059 pounds of prometone in 1983.

Pramitol 25E is diluted with water or oil and applied with high-volume hydraulic hose sprayers or portable sprayers in early spring to early summer. Foliage and the soil surface is sprayed at an effective application rate of 3,365 mg/m^2 prometone (active ingredient).

Pramitol 5PS is applied with portable spreaders to the soil surface before or after plant growth begins. The effective application rate of prometone (active ingredient) is between 1,232 and 4,930 mg/m^2, and application is timed to allow rainfall to move the chemicals into the plant root zone.

Fate and Distribution in the Environment

Soil

Prometone is more readily adsorbed on muck or clay soils than on soils of low clay or organic matter content (WSSA 1979).

Microbial activity probably accounts for the major breakdown of prometone in the soil. Soil microorganisms can utilize prometone as a source of energy and nitrogen. Prometone has negligible effects on soil microorganisms (WSSA 1979).

Photodecomposition and volatilization of prometone are not important means by which the herbicide is lost from soil.

Water

Since prometone is less tightly adsorbed to soil than atrizine and simazine, one would expect that slightly higher amounts of prometone might be present in runoff waters produced by rainfall shortly following herbicide application. Specific studies on prometone are not available, therefore reference is made to studies on atrizine and simazine, which are also triazine compounds.

In Iowa where large amounts of atrizine and simazine are used in corn production, those s-triazine herbicides are present in runoff, surface and groundwater (Brown 1978). Rainfall immediately after application results in parts per million levels of the s-triazines in runoff. Surface and groundwater concentrations are in the parts per billion range and decline from early summer through late summer due to the pattern of use of the herbicides.

Low to moderate mobility of s-triazines, including prometone, reduces the possibility of vertical leaching of simazine to groundwater (USDA 1978). It is not known how the less tightly adsorbed prometone will leach to groundwater.

Air

The fate of prometone in air has not been studied. Volatilization is not considered an important route of prometone loss from soil.

Chemical Toxicology in Animals and Humans

Acute Effects

Prometone has a low order of acute oral toxicity. The acute oral LD50 in rats is 2,980 mg/kg and in mice is 2,160 mg/kg (WSSA 1979).

In one method of interpreting toxicity data, it is presumed that acute (LD50) toxic doses for rat populations are similar to that for human populations. Given an LD50 of 2,980 mg/kg for rat populations, it is presumed that the LD50 dose of prometone (active ingredient) for a population of 165-pound humans would be 7.9 ounces (0.048 oz/lb).

Chronic Effects

No mortality was observed in rats given 400 mg/kg/day for six consecutive days of the week for four weeks (WSSA 1979).

Prometone was one of many chemicals tested for its ability to induce point mutations in eight strains of histidine-requiring mutants of bacteria (Salmonella typhimurium) (Anderson et al. 1972). Known mutagens were positive, but prometone and over 100 other herbicides were negative in these tests.

Potential Impact on Nontarget Organisms

Vegetation

Prometone is a nonselective preemergence and postemergence herbicide which can be used to control most annual and broadleaf weeds and certain perennial weeds. Combined with simazine, sodium chlorate, and sodium metaborate, a greater

variety of perennial weeds is controlled and the period of weed control lasts longer (Klingman and Ashton 1975).

Factors including absorption, translocation, and degradation may be related to prometone tolerance or resistance (Ashton and Crafts 1973).

Fish and Aquatic Organisms

Prometone has a low order of toxicity to fish. Spot (Leiostomus xanthurus) were not affected by a 48-hour exposure to 1 ppm prometone (Pimentel 1971). Oyster shell growth was not affected by a 96-hour exposure to 1 ppm prometone (Pimentel 1971). Pink shrimp were not affected by a 48-hour exposure to 1 ppm prometone (Pimentel 1971).

Wildlife

Testing in bobwhite quail and mallard ducks using diets containing prometone have shown the herbicide to have very low toxicity (WSSA 1979). The levels of prometone in the diet are proprietary information of Ciba-Geigy, and were not reported by WSSA (1979).

Livestock

The toxicity of prometone to cattle, sheep, and chickens has been studied by Palmer and Radeleff (1969). Cattle and sheep were more susceptible to orally-administered, water-diluted formulation than to capsules of prometone. Cattle were poisoned by 10 dosages of 10 mg/kg and sheep by 10 dosages of 25 mg/kg. Chickens dosed for 10 days at 25 mg/kg had a significantly reduced rate of weight gain. Signs of poisoning in cattle and sheep were anorexia (loss of appetite), diarrhea, and increased salivation. A sheep given 100 mg/kg for four days had petechiae (minute hemorrhagic spots) on the surface of the abomasal mucosa, hemorrhage in the small intestine, swollen and friable liver, and kidney congestion.

Application rates in excess of one pound/acre would make feed hazardous to cattle and rates in excess of three pounds/acre would make feed hazardous to sheep and chickens (Palmer and Radeleff 1969).

Hazard Assessment for Current Use Practice

Prometone is an effective preemergence and postemergence herbicide which is used by BPA in very small amounts in soil

sterilization operations at substations. Prometone in combination with simazine, sodium metaborate, and sodium chlorate (Pramitol 5PS) is an important soil sterilant which is applied to soil surface.

Based upon testing in laboratory animals, prometone has a low order of toxicity. As a result, CAUTION is the Human Hazard Signal Word displayed on herbicide labels.

Metabolic studies have shown that prometone does not accumulate in tissues, a finding similar to results obtained with atrazine and simazine.

Results of toxicological studies conducted by the manufacturer in support of the registration of prometone are proprietary, and have been reviewed by the Environmental Protection Agency. Full reports on the toxicology of prometone have not been published in the scientific literature.

Use of prometone as indicated on the pesticide label results in a low degree of hazard to personnel, the public, and the environment.

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SIMAZINE

Chemical Identification

Simazine is the common name for 2-chloro-4,6-bis(ethylamino)-s-triazine, an active component of Pramitol 5PS. Marketed by the Agricultural Division, Ciba-Geigy Corp. (Greensboro, North Carolina), Pramitol 5PS (EPA Reg. No. 100-479) is formulated as a pellet. The herbicide contains 95 percent active ingredients including simazine (0.75 percent), prometon (5.00 percent), sodium chlorate (40.00 percent), and sodium metaborate (50.00 percent). The latter ingredients are the subjects of other Background Statements (page A-117 for prometone and page A-131 for sodium chlorate and sodium metaborate).

Simazine is a white, crystalline solid which melts at 225°C to 227°C. The vapor pressure of simazine at 20°C is 6.1×10^{-9} mm Hg and 9.0×10^{-7} mm Hg at 50°C. Simazine is subject to decomposition by ultraviolet light (WSSA 1979). The solubility of simazine (ppm by weight at 20°C) in chloroform, methanol, petroleum ether, and water is 900, 400, 2, and 3.5, respectively.

Action in Vegetation

Simazine is registered for use in the selective weed control of many broadleaf and grass weeds in fruit crops including blueberries, caneberries, (blackberries, boysenberries, loganberries, and raspberries), cranberries, grapefruit, and oranges.

Simazine also may be used for nonselective weed control in noncroplands including industrial sites, highway medians and shoulders, railroad rights-of-way, lumberyards, petroleum tank farms, and noncrop areas on farms. Additional uses include nurseries, Christmas tree plantings, and shelter belts (Geigy Chemical Corp. 1970a).

Rapid absorption of simazine occurs through the roots, and the herbicide is readily translocated throughout the plant in the transpiration stream.

Simazine, like other s-triazine herbicides, inhibits plant growth as a consequence of its inhibition of photosynthesis (Ashton and Crafts, 1973). Foliar chlorosis precedes the death of the plant. Sublethal amounts of simazine may

stimulate growth and increase chlorophyll content. A dark green color of resistant crops has frequently been observed (Ashton and Crafts, 1973).

Replacement of the 2-chloro group with an hydroxy moiety and N-dealkylation are important steps in the degradation of simazine in plants. Roth (1957) described the rapid degradation of simazine. Sap from corn, a resistant species, was active in simazine degradation, but wheat sap, a sensitive species, did not degrade the herbicide. The degradation of simazine in higher plants and fungi has been summarized by Kearney (1969). The molecular fate of triazines in general has been reviewed by Ashton and Crafts (1973).

Utilization by BPA

Simazine is used by BPA to control weeds at substations. The herbicide is used as an active ingredient of Pramitol 5PS. BPA used on the average the equivalent of 609 pounds of simazine (active ingredient) per year since 1978, and proposes to use 459 pounds of simazine in 1983.

Pramitol 5PS is applied with portable spreaders to the soil surface before or after plant growth begins. The effective application rate of simazine (active ingredient) is between 168 and 683 mg/m^2, and application is timed to allow rainfall to move the chemicals into the plant root zone.

Chemical Fate and Distribution in the Environment

Soil

"Simazine is more readily adsorbed on muck or clay soils than in soils of low clay and organic matter content. The downward movement or leaching of simazine is limited by its low water solubility and adsorption to certain soil constituents. Tests have shown that for several months after application, the greatest portion will be found in the upper two inches of soil. It has little, if any, lateral movement in soil, but can be washed along with soil particles" (WSSA 1979).

Runoff from recently treated soils is the predominant means of movement of s-triazines from soil to water. Triplett et al. (1978) studied the movement of simazine from conventional and no-tillage corn watersheds. Highest concentrations of simazine (1.2 ppm) were measured in runoff which occurred shortly after application. Later runoff contained

lower amounts. A maximum of 6 percent of the applied herbicide was transported from the test plots and the average for all watersheds was 2 percent. No-tillage areas had lower runoff and lower simazine transport than conventional corn plots.

Low to moderate mobility of s-triazines including simazine reduces the possibility of vertical leaching of simazine to groundwater (USDA 1978).

Simazine will persist longer in fine textured soils than in sandy soils. Cold temperatures and dry conditions also favor persistence. These conditions do not generally favor chemical and microbial breakdown of simazine (or other organic herbicides) (WSSA 1979).

Microbial activity in soils accounts for significant amounts of simazine degradation (WSSA 1979). Sterilized soils degrade simazine less readily than nonsterilized soils (Brown 1978). Soil organisms, including fungi and bacteria, that are active in simazine degradation have been tabulated (Brown 1978). Chemical and microbial activity are important in the formation of hydroxysimazine. Simazine applied at normal field rates did not reduce total microfloral activity as judged from the CO_2 production by the treated soil (references in Brown 1978).

Water

Simazine has been measured in surface waters in Iowa where it is extensively used in corn production. Several examples are cited in a review (Brown 1978).

Air

Volatilization is of negligible importance in the reduction of simazine levels in soil (WSSA 1979), therefore little simazine will occur in the atmosphere.

Chemical Toxicology in Animals and Humans

Acute Effects

Simazine has a low order or acute oral toxicity. The acute oral LD50 of simazine is greater than 5,000 mg/kg in rats, mice, rabbits, chickens, and pigeons (Geigy Chemical Corp. 1970b).

The acute dermal LD50 of simazine in rabbits (single exposure) is greater than 10 grams/kg. In a 21-day repeated

dermal exposure study, the LD50 was 2 grams/kg (Geigy Chemical Corp. 1970b).

No substantial skin or eye irritation has been reported from either experimental or commercial use (USDA 1974-75).

Low inhalation hazard is present. "No deaths or signs of toxicological or pharmacological effects resulted from exposing groups of rats for one hour to a dust aerosol of simazine 80W. Aerosol concentrations ranged from 1.8 to 4.9 mg/liter of atmosphere" (USDA 1974-75).

In one method of interpreting toxicity data, it is presumed that acute (LD50) toxic doses for rat populations are similar to that for human populations. Given an LD50 of 5,000 mg/kg for rat populations, it is presumed that the LD50 dose of simazine (active ingredient) for a population of 165-pound humans would be 13.2 ounces (0.080 oz/lb).

Chronic Effects

Two-year chronic oral feeding studies produced no gross or microscopic signs of systemic toxicity. Male and female rats were given daily dietary levels of up to 100 ppm simazine (as a 50 percent wettable powder formulation) (Geigy Chemical Corp. 1970b).

Sheep fed up to 25 mg/kg for five weeks remained normal (Geigy Chemical Corp. 1970b).

Complete results of chronic toxicity testing are proprietary information retained by the manufacturer and the Environmental Protection Agency.

Simazine was among many chemicals tested for ability to induce point mutations in eight strains of histidine-requiring mutants of bacteria (Salmonella typhimurium) (Anderson et al. 1972). Known mutagens were positive, but simazine and over 100 other herbicides were negative in these tests.

Potential Impact on Nontarget Organisms

Vegetation

Simazine is an important selective herbicide in vegetation management. Simazine is registered for use on more crops than any other triazine. Its effective use in nursery

plants and Christmas tree plantations requires lower application rates than those required when simazine is used as a nonselective herbicide on noncroplands.

Corn and other higher plants may tolerate simazine due to rapid degradation of the herbicide. Other higher plants may tolerate simazine exposures due to their failure to absorb and translocate the herbicide. Tolerant white pine (Pinus strobus) contained only one-third as much simazine in its needles as sensitive red pine (Pinus resinosa) (Freeman et al. 1964).

Herbicide use from 1958 to 1968 resulted in development of atrazine and simazine resistance in common groundsel in the state of Washington (Ryan 1970).

Fish and Aquatic Organisms

Simazine has a low order of toxicity to fish. Pimentel (1971) listed the LD50s for three species in six separate studies. Rainbow trout LC50s were 68 ppm (24-hour) and 5 and 56 ppm in two separate 96-hour studies. The LC50 in bluegills was 130 (24-hour) and 118 (48-hour). In striped bass, the 24-hour LC50 was 0.60 ppm.

Simazine persisted in fish for only short intervals. Half of the herbicide was lost in less than three days (Macek 1969).

Pimentel (1971) further reported toxicity measurements in other organisms. The 48-hour LC50 for stoneflies and amphipods (Gammarus lacustris) were 50 and 21 ppm simazine, respectively. The 24-hour LC50 for a second study was 30 ppm. Some arthropods are more sensitive to simazine. Concentrations of 0.5 to 10 ppm reduced populations of mayflies, mosquitoes, biting midges, damselfly nymphs, water beetles, aquatic worms, leeches, and snails (Walker, 1962; cited in Pimentel 1971).

Wildlife

Consistent with results of toxicity testing in rodents, simazine has a low order of activity in wildlife. Diets containing simazine were fed for five days to two-week-old mallard ducks, pheasants, and coturnix quail. No toxicity was observed after the eight-day test period at dietary levels of greater than 5,000 ppm simazine (Pimentel 1971).

Livestock

The toxicity of simazine to cattle, sheep, and chickens has been studied by Palmer and Radeleff (1969). Cattle orally given a water-diluted formulation at a dosage of 25 mg/kg were poisoned after three and ten doses. One sheep was poisoned after 17 doses of 50 mg/kg and died after 31 treatments. A second sheep was poisoned after 10 doses but survived with an 18 percent weight loss. The weight gain of chickens was reduced by 10 doses of 50 mg/kg. Signs of poisoning in cattle and sheep were anorexia (loss of appetite), weight loss, muscular spasms, and dyspnea (labored breathing). In severe poisoning, weakness and uncoordinated gait were observed. At necropsy, lung and kidney congestion, swollen, friable, and often light-brown liver, and petechiae (minute hemorrhagic spots) on the surface of the epicardium were reported. A chicken had an enlarged, congested liver and congestion of intestinal mucosa.

Application rates in excess of 3, 5, and 9.6 pounds/acre would make feed hazardous to cattle, sheep, and chickens, respectively (Palmer and Radeleff 1969).

Hazard Assessment for Current Use Practices

Simazine is one of the active ingredients in Pramitol 5PS, an herbicide containing 95 percent active ingredients including 0.75 percent simazine. Most phytotoxic effects of the herbicide are due to the effective soil sterilization activity of the other components of the mixture. Simazine is added to the product to improve control of certain grasses and broadleaf weeds.

Based upon testing in laboratory animals, simazine has a low order of toxicity in animals. Acute tests using Pramitol 5PS have also shown it to have low toxicity. As a result, the product is labelled CAUTION in accordance with the Human Hazard Signal Word system.

Laboratory tests have shown simazine to be free of mutagenic activity. Rapid simazine metabolism and excretion in animals reduces the possibility of chronic effects. Long-term, low-level exposures are extremely unlikely as a result of use of simazine in BPA vegetation management.

Simazine is moderately stable and persistent in treated soils. Phytotoxic quantities of simazine may persist more than a year. Simazine is adsorbed by soil organic matter. Microbial and chemical degradation are much more important than volatility and photodecomposition in the reduction of

soil herbicide. The toxicity of sodium chlorate and metaborate to soil microorganisms will prolong the effectiveness of simazine. Losses from soil via leaching will be negligible due to the adsorption and the low water solubility of simazine.

Exposure of nontarget organisms is unlikely in soil sterilization operations. Most plants would be sensitive to the phytotoxic effects of Pramitol 5PS. Aquatic organisms, wildlife, and livestock are unlikely to be exposed to simazine.

Use of simazine as indicated on the pesticide label results in a low degree of hazard to personnel, the public, and the environment.

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SODIUM METABORATE AND SODIUM CHLORATE

Chemical Identification

Sodium metaborate ($NA_2B_2O_4.4H_2O$) and sodium chlorate ($NaClO_3$) are active ingredients in Oxy Ureabor (Occidental Chemical Company, Houston, Texas). The EPA registration number of Oxy Ureabor is 10659-51. Oxy Ureabor contains bromacil (1.5 percent) in addition to sodium metaborate (66.5 percent) and sodium chlorate (30.0 percent). Bromacil is the subject of a separate Background Statement (page A-34). Sodium metaborate and sodium chlorate are also active ingredients in Pramitol 5PS (EPA Reg. No. 100-479). Pramitol 5PS also contains prometone and simazine as active ingredients. Prometone and simazine are the subjects of separate Background Statements (page A-117 and page A-123, respectively).

Sodium metaborate is an odorless, white solid which is highly soluble in water (48 grams/100 ml). Sodium chlorate is an odorless, pale yellow to white solid. At 0°C, 79 grams are soluble in 100 ml water and at 100°C, 230 grams are soluble in 100 ml water.

Action in Vegetation

Boron is an essential minor element for plant growth, but in excessive amounts boron and borates are toxic to plants and act as a soil sterilant.

Sodium borates are absorbed principally by roots and are translocated to all parts of the plant. Borates accumulate in leaves and cause plant desiccation, which is initially evident as leaf burn and necrosis of leaf margins. Borate is most effective on young, tender plants (Klingman and Ashton 1975).

Sodium chlorate is generally used as a sterilant to kill all vegetation. The herbicide is absorbed rapidly through roots and leaves. Chlorate moves rapidly from the roots upward through the xylem. Its toxic action in plants is related to depletion of the plant's food reserves, to temporary increases in rate of respiration, and to decreases in catalase activity (Klingman and Ashton 1975).

These inorganic substances have been extensively used singly and in combination with other herbicides. Their precise mechanism of action remains unknown. Sodium chlorate toxicity may result from the reduction of chlorate to chlorite, catalyzed by nitrate reductase (Murphy and Imbrie 1981).

Utilization by BPA

Sodium metaborate and sodium chlorate are used by BPA as formulated in Oxy Ureabor and Pramitol 5PS. The formulations are used at substations to control weeds. BPA used on the average the equivalent of 45,398 pounds of sodium chlorate and 67,010 pounds of sodium metaborate per year since 1978. BPA proposes to use the equivalent of 24,473 and 30,591 pounds of sodium chlorate and sodium metaborate, respectively, in 1983.

Oxy Ureabor is applied with portable spreaders or as an aqueous mixture with portable sprayers. Application to the soil surface is made during the growing season at an effective application rate of sodium chlorate (active ingredient) between 7,280 and 43,792 mg/m^2, and an effective application rate of sodium metaborate (active ingredient) between 12,208 and 48,832 mg/m^2.

Pramitol 5PS is applied with portable spreaders to the soil surface before or after plant growth begins. The effective application rate of sodium chlorate and sodium metaborate (active ingredients) is between 7,280 and 43,792 mg/m^2 and between 16,240 and 47,104 mg/m^2, respectively. Application is timed to allow rainfall to move the chemicals into the plant roots zone.

Chemical Distribution and Fate in the Environment

Soil and Water

Uniform soil applications of dry granules or spray are important to the herbicidal action of Oxy Ureabor. Sodium metaborate and sodium chlorate are carried into the root zone by rainfall.

Leaching quickly removes sodium chlorate from soil (Seely et al. 1948). Soil microorganisms decompose chlorates to chlorides which lack the phytotoxicity of the parent compound. This decomposition is most rapid in moist soils above 70°F. With low rainfall, chlorate may remain toxic for five years or longer. Under humid or wet conditions,

toxicity may disappear in a year or less in heavy soils and more rapidly in sandy soils (Klingman and Ashton 1975).

Sodium borate is also carried into the soil by rainfall. In warm moist soils, borate usually remains effective for about one year. In dry or in frozen soils, sterilant effects will persist for several years (Klingman and Ashton 1975).

Air

These substances are nonvolatile and not degraded by light.

Chemical Toxicology in Animals and Humans

Acute

Sodium metaborate and sodium chlorate have low orders of toxicity. The oral LD50 of sodium metaborate in the rat is 2,330 mg/kg. The oral LD50 of sodium chlorate in the rat is 5,000 mg/kg.

The acute oral LD50 of Oxy Ureabor in male rats is 2,710 mg/kg. The acute dermal LD50 in rabbits is greater than 10,000 mg/kg (Occidental Chemical Company 1981).

Oxy Ureabor is a mild skin irritant. If applied as a wet paste for several hours, it causes erythema (inflammation) and edema with focal gray discolored areas which may proceed to superficial blisters or shallow ulcers. Based upon this property, the herbicide is labelled "DANGER" (Human Health Signal Word), and emergency treatment is specified on the label.

In one method of interpreting toxicity data, it is presumed that acute (LD50) toxic doses for rat populations are similar to that for human populations. Given an LD50 of 5,000 mg/kg for rat populations, it is presumed that the LD50 dose of sodium chlorate (active ingredient) for a population of 165-pound humans would be 13.2 ounces (0.08 oz/lb). Given an LD50 of 2,330 mg/kg for rat populations, it is presumed that the LD50 dose of sodium metaborate (active ingredient) for a population of 165-pound humans would be 6.2 ounces (0.037 oz/lb).

Chronic

Chronic studies are lacking in the literature. Cases of chronic chlorate toxicity are unknown (WSSA 1979).

Potential Impact on Nontarget Organisms

Vegetation

Sodium borate and sodium metaborate are nonselective herbicides with respect to their toxicity in plants. Young plants are more sensitive than mature plants to their toxic effects.

Fish

The 24-hour LC50 for rainbow trout to borax is 2,800 ppm (Pimentel 1971). Toxicity tests with sodium chlorate have not been reported.

Wildlife

Toxicity testing in wildlife has not been conducted. Based upon the very low order of toxicity in laboratory animals, toxicity in wildlife also would be expected to be low.

Livestock

Special studies in livestock have not been reported.

Hazard Assessment for Current Use Practice

Sodium borate and sodium metaborate are inorganic herbicides that have been used since before the introduction of organic herbicides in the 1940s. These substances are used as soil sterilants for the control of weeds at substations.

Oxy Ureabor is labelled "DANGER" (Human Health Signal Word) as a result of its corrosiveness. The label indicates the following statement of practical treatment: "In case of contact with eyes, flush with water for 15 minutes. Get medical attention. In case of contact with skin, wash with plenty of soap and water. Get medical attention if irritation persists."

Both of these substances have a low order of acute toxicity in laboratory animals.

Sodium borate and sodium metaborate are persistent as inorganic chemicals. They are moved into the soil by rainfall and remain effective about one year. Boron is toxic to most soil microflora, and as a result, the

persistence of organic substances (such as bromacil in Oxy Ureabor) is prolonged due to diminished microbial degradation. Borate is more persistent than chlorate in most soils.

Use of sodium chlorate and sodium metaborate as specified on the pesticide label results in a low degree of hazard to personnel, the public, and the environment.

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TEBUTHIURON

Chemical Identification

Tebuthiuron is the common name for N-[5-(1,1-dimethylethyl)-1,3,4-thiadiazol-2-yl]-N,N'-dimethylurea. Spike is the trade name for tebuthiuron herbicides that are marketed by Elanco Products Company (Divisions of Eli Lilly and Company, Indianapolis, Indiana).

Tebuthiuron is a urea derivative which is a colorless, odorless solid at room temperature. It melts at 161.5 to 164°C. The vapor pressure of tebuthiuron is 2×10^{-6} mm Hg at 25°C. The solubility of tebuthiuron is 6 grams/100 ml or more in methyl cellosolve, acetonitrile, acetone, methanol, and chloroform. The limit of water solubility is 0.24 g/100 ml.

Spike 1G (EPA Reg. No. 1471-104) and Spike 5G (EPA Reg. No. 1471-103) are hard, grayish materials with the appearance of finely crushed stone containing 1 percent and 5 percent tebuthiuron, respectively. They have faint, kerosene-like odors. Spike 1G and 5G are intended for total vegetation control on noncroplands.

Spike 80W (EPA Reg. No. 1471-97) is a very fine, off-white wettable powder with a mild odor. This dispersible formulation must be kept agitated at all times to assure uniform application. Spike 80W is stable under normal conditions of storage and use. The product safety data sheet (Elanco 1980) includes the following warning: "The explosive potential of Spike 80W as an airborne dust is rated as severe. The minimum ignition temperature of a dust cloud is 948°F (509°C). Spills or accumulations of dust should be cleaned up immediately." Spike 1G and Spike 5G have no unusual fire or explosion hazards.

Action in Vegetation

Tebuthiuron is toxic to annual and perennial grasses and broadleaf weeds. Soil moisture promotes the rapid absorption of tebuthiuron requiring that material be applied before or early in the rainy season.

Tebuthiuron is readily absorbed from soil through plant roots and less readily through foliage. Translocation to leaves is rapid. Tebuthiuron inhibits photosynthesis,

causing leaf senescence and subsequent defoliation. Several defoliation cycles following heavy rainfalls may occur before plant death (McNeil et al. n.d.).

Utilization by BPA

Tebuthiuron is used to control weeds at substations and around transmission towers and poles. BPA used 185 and 1,386 pounds of tebuthiuron (active ingredient) in 1980 and 1981, respectively. It was not used by BPA during 1978 and 1979. The equivalent of 1,714 pounds of active ingredient is proposed for use in 1983.

Spike 1G and Spike 5G are granular forms applied to the soil surface with portable spreaders shortly before or at the time of new plant growth. Application of the product is made at a rate which results in an effective application rate of tebuthiuron (active ingredient) between 448 and 1,792 mg/m^2.

Spike 80W is diluted with water and applied to the soil surface with portable sprayers at any time of the year. Effective application rate of tebuthiuron is between 134 and 1,792 mg/m^2.

Chemical Fate and Distribution in the Environment

Soil

Volatilization and photodecomposition are of negligible importance in the loss of tebuthiuron from soil (WSSA 1979). Although microbial degradation of tebuthiuron occurs, the reduction of soil residues probably does not involve soil microbes to an appreciable extent.

Tebuthiuron has a half-life in soil of 12 to 15 months in areas receiving 40 to 60 inches of annual rainfall. The half-life is considerably longer in areas of low rainfall. Muck and other high organic soils also prolong the persistence of tebuthiuron. Tebuthiuron and metabolites are seldom detected below the top 12 inches of soil. Little or no lateral movement occurs (WSSA 1979).

Water

The concentration of tebuthiuron was measured in surface runoff water from watersheds where the herbicide was applied in spray or pelleted form. Pelleted tebuthiuron was applied at a rate of 2.24 kg/ha to a 1.3 ha rangeland watershed. A

2.8 cm rainfall two days after application produced 0.94 cm runoff which contained an average of 2.2 ppm tebuthiuron. Each subsequent runoff contained less herbicide and, after three months, the concentration was 0.05 ppm. None was detectable one year after application. Spray at 1.12 kg/ha to a small plot which received simulated rainfall resulted in runoff concentrations of 0.04 ppm after four months. On 0.6 ha plots mean tebuthiuron concentrations in the first runoff (two months after application) were 0.50 ppm or less (Bovey et al. n.d. cited in U.S. Forest Service 1981).

Air

Studies of the fate of tebuthiuron in air are not available.

Chemical Toxicology in Animals and Humans

Acute Effects

The acute oral LD50s of tebuthiuron to mice, rats, and rabbits are 579, 644, and 286 mg/kg (Todd et al. 1974), respectively. Similarly, no deaths were obtained in cats (LD50 greater than 200 mg/kg) and dogs (LD50 greater than 500 mg/kg). This low order of acute toxicity is similar to that observed in studies of monuron and diuron (see other Background Statements, page A-89 and page A-67, respectively).

Tebuthiuron caused no irritation of the cornea or iris of rabbit eyes (71 mg/eye) but there was a slight transient hyperemia (excess blood) (Todd et al. 1974). All eyes were normal at the end of the seven-day test. Tebuthiuron did not cause dermal irritation (200 mg/kg exposure for 24 hours) during a 14-day observation peiod. No evidence of skin sensitization was obtained in studies of guinea pigs treated three times per week for three weeks then challenged later with the herbicide (Todd et al. 1974). Persons handling tebuthiuron are exposed to a low order of hazard.

In one method of interpreting toxicity data, it is presumed that acute (LD50) toxic doses for rat populations are similar to that for human populations. Given an LD50 of 644 mg/kg for rat populations, it is presumed that the LD50 dose of tebuthiuron (active ingredient) for a population of 165-pound humans would be 1.7 ounces (0.010 oz/lb).

Chronic Effects

Male and female rats were fed 0, 400, 1,000 and 2,500 ppm tebuthiuron in their diets for three months. A dose-dependent reduction in the rate of body weight gain was observed. No effects on either blood or clinical chemistry were observed. There were differences in absolute and relative organ weights related to the smaller body weights of the rats at the high dietary levels (Todd et al. 1974). All rats receiving 2,500 ppm tebuthiuron exhibited diffuse vacuolization of the pancreatic acinar cells. The change was moderate to severe but not associated with either necrosis or an inflammatory response. This cellular change may interfere with the production and release of digestive enzymes, and could be related to the reduced weight gain of the rats (Todd et al. 1974).

Dogs were given daily oral dosages of 0, 12.5, 25, and 50 mg/kg tebuthiuron. The treated dogs ate less than controls and a slight weight loss was reported in two of four dogs at the highest dosage. No treatment-related effects on blood and clinical chemistry, organ weights, and tissue histopathology were observed. Pancreatic vacuolization was not observed in any of the treated dogs (Todd et al. 1974).

Lifetime feeding and carcinogenicity studies have been completed in rats and mice and are being evaluated (WSSA 1979).

A teratology study produced no significant effects in the offspring of rats fed diets containing 0, 600, 1,200 or 1,800 ppm tebuthiuron. No effects on several reproduction indices were observed in these studies (Todd et al. 1974).

Orally dosed mice, rats, dogs, and ducks readily absorb tebuthiuron. The compound was extensively metabolized and excreted in urine by mice, rats, and dogs, and in urine and feces of ducks. N-demethylation was the most important degradation process. No accumulation of tebuthiuron or metabolites was observed in the animals (WSSA 1979).

Potential Impact on Nontarget Organisms

Vegetation

A prominent warning on the labels of Spike 80W, Spike 1G, and Spike 5G concerns the potency of tebuthiuron: "...is intended for total vegetation control. It is an extremely active herbicide which will kill trees, shrubs, and other forms of desirable vegetation having roots extending into

the treated area. Feeder roots of many species of desirable vegetation extend many feet beyond the drip line of the branches, and a very small amount of Spike in contact with one feeder root of a tree, shrub, or other desirable vegetation may cause serious injury or death to the entire plant."

Fish and Aquatic Organisms

The median threshold limit values for trout and bluegill are 144 and 112 ppm, respectively (time not specified; WSSA 1979).

Wildlife

The acute oral toxicity of tebuthiuron to quail and ducks is of a low order of magnitude. No mortality in either species was observed at 500 mg/kg.

Livestock

The "safe level" after one month of feeding in chickens is 1,000 ppm (Todd et al. 1974). Studies in livestock have not been reported. Tebuthiuron labels state that livestock should not be allowed to graze treated areas nor should they be fed forage from treated areas.

Hazard Assessment for Current Use Practice

Tebuthiuron is an effective herbicide which can be used for control of weeds at substations and around utility poles. This herbicide is used by BPA extensively at substations.

Tebuthiuron is a urea-based herbicide with a low order of toxicity in animals. Based upon the results of toxicity testing, the Human Hazard Signal Word "CAUTION" is printed on the herbicide label.

Tebuthiuron has low chronic toxicity. Like other urea-based herbicides, tebuthiuron is rapidly metabolized and excreted from test animals.

The environmental fate of tebuthiuron has been evaluated under a variety of laboratory and field conditions. Tebuthiuron is relatively persistent in treated soils. Volatility, photodecomposition, and chemical and/or biological degradation are of minor importance in the reduction of soil tebuthiuron.

More important are the amounts of rainfall and the extent of soil adsorption. The herbicide will remain in the upper soil levels (0-12 inches) and extreme care must be exercised to avoid the root zone of desirable trees and shrubs.

The amounts of tebuthiuron that might be present in surface runoff are well below those that could cause toxicity in fish. Wildlife and livestock are not sensitive to tebuthiuron consistent with its low order of toxicity in laboratory animals.

Use of tebuthiuron as prescribed on the herbicide label presents low hazard to personnel, the public, and the environment.

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Chemical Identification

2,4-D is the common name for 2,4-dichlorophenoxyacetic acid (and here will be used generically to describe various 2,4-D formulations).

2,4-D is a white crystalline solid which melts at 140°C to 141°C. The vapor pressure of 2,4-D is 0.4 mm Hg at 160°C. At 25°C, 85, 130, 27, 0.58 g are soluble in 100 g acetone, ethanol (95 percent), ethyl ether, and xylene, respectively. 2,4-D has low water solubility, 0.09 g/100 g water (900 ppm). The salts of 2,4-D (sodium, lithium, amine) are very soluble in water.

The concentration of 2,4-D and other phenoxy herbicide formulations is expressed in acid equivalents per gallon. Recommendations for rate of application are made on that basis. An acid equivalent is the amount of a formulation that can be converted to 2,4-D.

Amine salts are the most commonly used form of 2,4-D. Formula 40 (EPA Reg. No. 464-1-AA; The Dow Chemical Company) contains the dimethylamine salt of 2,4-D, a white crystalline solid which melts (with decomposition) at 85°C to 87°C. The salt is extremely soluble in water (300 g dissolves in 100 g water).

Ester formulations of 2,4-D are also used by BPA. The propylene glycol butyl ether ester of 2,4-D is marketed as Esteron 99 (EPA Reg. No. 464-201-AA; The Dow Chemical Company). This ester is a colorless liquid which is essentially insoluble in water. This long chain ether ester is of especially low volatility, which minimizes the hazard of fumes and vapors. Weedone 170 Woody Plant Herbicide (EPA Reg. No. 264-222 ZB; Union Carbide) is a concentrated combination of the butoxyethanol esters of 2,4-D and 2,4-DP (2,4-dichlorophenoxypropanoic acid).

Other herbicide formulations containing 2,4-D are used by BPA. Tordon 101 Mixture Weed and Brush Killer (EPA Reg. No. 464-306; The Dow Chemical Company) contains the triisopropanolamine salts of 2,4-D and picloram (the subject of a separate Background Statement, page A-102). Tordon 101 is an important herbicide in BPA vegetation management. Banvel 520 (EPA Reg. No. 876-168 AA) and Banvel 720 (EPA Reg. No.

876-177 AA) contain 2,4-D and dicamba as active ingredients. Dicamba is the subject of a separate Background Statement (page A-47). Banvel 520 contains the isooctyl ester of 2,4-D and Banvel 720 contains the dimethylamine salt of 2,4-D. Banvel 720 also is an important herbicide in BPA vegetation management.

2,4-DP (also known as dichlorprop) is the propanoic acid derivative of 2,4-D used for brush control in vegetation management. The toxicity and environmental behavior of 2,4-D and 2,4-DP are similar; therefore, 2,4-DP will not be the subject of a separate Background Statement. 2,4-DP is especially effective for woody plant control and for management of 2,4-D resistant plant species.

Much of the controversy related to the use of herbicides in vegetation management is related to the chemical synthesis of phenoxy herbicides and toxicological properties of chlorodibenzodioxin impurities. 2,4,5-T (2,4,5-trichlorophenoxyacetic acid) is synthesized from 2,4,5-trichlorophenol and can contain part per million amounts of an extremely toxic contaminant, 2,3,6,7-tetrachlorodibenzo-p-dioxin (TCDD).

Chlorodibenzodioxins other than TCDD are of less concern because of low toxicity. Schwetz et al. (1973) reported that 2,4-dichlorodibenzo-p-dioxin and octachlorodibenzo-p-dioxin have low toxicity, whereas TCCD was extremely toxic. Low dosages of TCCD (0.0005 to 0.001 mg/kg/day) were toxic to rats, whereas 1,2,3,4-tetrachlorodibenzo-p-dioxin 2,7-dichlorodibenzo-p-dioxin, 2,3-dichlorodibenzo-p-dioxin, and 2-chlorodibenzo-p-dioxin at dosages of up to 2 mg/kg/day had little or no effect (Khera and Ruddick 1973).

2,4-D is synthesized from 2,4-dichlorophenol and, therefore, does not contain TCDD (see Bovey and Young 1980). Three other chlorodibenzodioxins of low toxicity have been found in 2,4-D manufactured in Canada (Cochrane et al. 1980). Analysis of 30 U.S. samples of 2,4-D revealed 2,7-dichlorodibenzo-p-dioxin in three formulations. No sample contained more than 60 ppb of 2,7-dichlorodibenzo-p-dioxin, which does not represent a toxicologic concern (Newton and Dost 1981). As part of the National Cancer Institute bioassay program, a two-year feeding study was conducted. Male and female rats and mice were fed 10,000 ppm (10,000,000 ppb) 2,7-dichlorodibenzo-p-dioxin. A "suggestion" of carcinogenic activity was found in male rats but not in female rats or mice. The panel of the National Cancer Institute concluded that 2,7-dichlorodibenzo-p-dioxin was not a carcinogen.

Action in Vegetation

The 2,4-D herbicides are members of the phenoxy herbicides group which is the most extensively used set of chemicals in vegetation management. A very important factor to consider in their use is the selectivity of phenoxy herbicides for broadleaf plants (Ashton and Crafts 1973). 2,4-D is effective in control of annual and perennial forbs and woody plants (shrubs, hardwoods, and conifers) on croplands.

2,4-D is more active as a foliar spray than as a soil application. Both upper and lower leaf surfaces absorb the herbicide and lower leaf surfaces are most readily penetrated. There is a direct relationship between the thickness of the plant cuticle (waxy outer layer) and the species sensitivity to phenoxy herbicides (Bovey and Young 1980). Following foliar absorption, 2,4-D is translocated within the phloem, probably moving with photosynthesis. Following root absorption, 2,4-D may move upward in the transpiration stream (WSSA 1979). Plants generally gain tolerance to 2,4-D with age.

The phenoxy herbicides are growth regulators with hormone (auxin)-like activity. Low levels of exposure promote growth responses in parts of the plant distant from the point of application. Further, foliar applications of 2,4-D can be used to kill root systems of perennial weeds. These observations indicate that 2,4-D is readily translocated and is phytotoxic in small amounts.

2,4-D causes abnormal plant growth and affects respiration, food reserves, and cell division (WSSA 1979). Primary toxic effects of 2,4-D in plants include the following: (1) twisting or bending of stems and leaves resulting from differential growth, (2) thickening of leaves and stems, and (3) cessation of growth and cell death.

The degradation of 2,4-D by higher plants and microorganisms has been reviewed (Loos 1975). Side chain degradation, dechlorination, hydroxylation, and ring opening are reactions which reduce the phytotoxicity of 2,4-D and other phenoxy herbicides.

The organic amine salts and esters of 2,4-D are readily converted to the free acid in plants. That conversion is necessary to the expression of phytotoxicity. An additional critical conversion is that of 2,4-DB to 2,4-D in susceptible plants (Loos 1975).

Utilization by BPA

The 2,4-D herbicides (including 2,4-DP) are used extensively by BPA to control forbs and woody plants (shrubs and trees) beneath transmission lines and towers, around poles, and at substations. Ground-based and aerial applications are used. BPA used on the average the equivalent of 30,516 pounds of 2,4-D and 473 pounds of 2,4-DP (active ingredients) per year since 1978, and proposes to use 1,954 pounds of 2,4-D and 0 pounds of 2,4-DP in 1983.

Formula 40 is either used as the formulated product or is diluted with water and applied to foliage and stems. Application can be made as a broadcast spray at a dilution and effective application rate of 224 mg/m^2 2,4-D amine (active ingredient). Application is also made as a spot (directed) spray (0.25 pint product in three gallons of water) which thoroughly wets all foliage, or as single-stem treatment which injects 1 to 2 ml undiluted product to each notch or injection point into the cambium around the circumference near the base of susceptible trees. Spray operations occur during warm weather when forbs and shrubs actively grow, whereas single-stem treatment occurs during any season, except maples are not treated during the spring sap flow.

Esteron 99 is diluted with water and broadcast by air or ground-based equipment to foliage and stems when alder trees are actively growing. The effective broadcast application rate of 2,4-D ester (active ingredient) is 448 mg/m^2. The formulation may also be diluted with water and applied as a directed spray which thoroughly wets all weed foliage.

Weedone 170 is diluted with water, or oil, or an oil and water mixture and applied as a ground-based broadcast spray, spot spray, or single-stem treatment. If broadcast, only water is used and the effective application rate of 2,4-D ester and 2,4-DP (active ingredients) is between 450 and 2,000 mg/m^2 for each. Application of spot sprays is made to thoroughly wet target vegetation. Single-stem treatment involves drenching cut stumps or pouring the diluted formula into cut surfaces near the base of the tree.

Tordon 101 is used to control weeds and woody plants on rights-of-way and at substations. The herbicide is applied as a high-volume, ground-based broadcast spray, as a directed (selective) spray, as an aerial treatment broadcast by a helicopter, or with single-stem and cut-stump treatment devices. Application is made after foliage is well developed, but while weeds and brush are actively growing. Dilution (with water) and broadcast application rates

provide an effective application rate of picloram (active ingredient) between 56 and 168 mg/m^2, and an effective application rate of 2,4-D amine (active ingredient) between 224 and 673 mg/m^2. When applied directly to single stems or cut stumps, dilution yields 60 and 240 grams/liter picloram and 2,4-D, respectively.

Banvel 520 is diluted with oil and applied with high volume hydraulic hose sprayers to brush and weeds during the dormant season. The formulation includes 2,4-D ester and dicamba as active ingredients. Vegetation is sprayed until runoff occurs on the basal part of the stem.

Banvel 720 is diluted with water and broadcast by air or ground equipment after leaves are fully developed and until three weeks before frost. The formulation contains 2,4-D amine and dicamba as active ingredients. Dilution and application rates provide an effective application rate of 2,4-D amine and dicamba (active ingredients) between 224 and 672 mg/m^2 and between 112 and 336 mg/m^2, respectively. Banvel 720 is also diluted with an equal quantity of water and applied to cut surfaces at any time of the year. Application of 0.5 to 1 ml of solution is made to each notch or injection point.

Chemical Fate and Distribution in the Environment

Soil

2,4-D is rapidly inactivated in moist soil with half-lives usually being measured in days to weeks (except under arid conditions where 2,4-D persists for longer periods). The herbicide does not accumulate or persist in phytotoxic amounts from one year to the next.

Soil microorganisms are very active in the degradation of soil residues of 2,4-D. By 1977, over 340 research papers had been published on microbial interactions with the phenoxy herbicides (Bovey and Young 1980). Some soil microorganisms are susceptible to phenoxy herbicides at concentrations of 50 ppm; however, most are tolerant. Concentrations of 2,4-D at 100 to 200 times the amount normally used for weed control usually have no appreciable effect on soil populations of bacteria, fungi, and actinomycetes. Rates of breakdown in moist loam soils would be one to three months when application rates are 4.5 to 61.6 kg/ha. In general, conditions which favor the growth of soil microorganisms (moisture, warmth, organic matter) likewise promote the rapid degradation of 2,4-D.

The molecular fate of 2,4-D has been extensively studied in soils (Loos 1975). Arthrobacter (a bacterium) enzymatically converts 2,4-D to 2,4-dichlorophenol and other chlorophenols. The phenols are hydroxylated to yield catechols which are further metabolized to yield butenolides and chloromuconic acid. Ultimate products of 2,4-D are chloride ion, acetate, and dicarboxylic acid. It is an important finding that chlorophenols are not the end products of 2,4-D degradation.

2,4-D may be degraded by chemical processes in soil in the absence of living organisms. Oxidation, reduction, and hydrolysis are known to occur. For example, isopropyl and n-butyl esters of 2,4-D were hydrolyzed in moist soil within 24 hours (Bovey and Young 1980).

Herbicide applied to plant and soil surfaces is subject to decomposition by sunlight under laboratory and field conditions. 2,4-dichlorophenol and mono dechlorinated hydroxy derivatives are formed. The ultimate degradation products are polymeric humic acids, substances found to occur widely in the organic fraction of soil (Bovey and Young 1980; Crosby and Tutass 1966).

Volatility of phenoxy herbicides can represent a pathway of loss from soil and plant surfaces. Baur and Bovey (1974) found that losses of more than 50 percent per day occurred when dry preparations were exposed to temperatures up to 60°C. Soil surface temperatures could reach 60°C under summer conditions. The ester formulations of 2,4-D are more volatile than other phenoxys used by BPA (see Chemical Identification section).

Soil adsorption is not as important as is microbial degradation in reducing soil concentrations of 2,4-D. In soil, 2,4-D exists as a negatively charged chemical or as an undissociated acid. Soils are generally negatively charged also, hence, the extent of adsorption is small. Organic matter, iron, and aluminum will enhance soil adsorption. Phenoxy herbicides are usually found in the top layers (0 to 6 inches) of soil (Bovey and Young 1980).

Water

2,4-D may be found in low concentrations in water as a result of vegetation management operations. Surface runoff is a possible means of movement of 2,4-D from treated areas into water. Bovey and Young (1980) reviewed the occurrence of 2,4-D in surface runoff water in cultivated and pasture lands, irrigation water, impounded water, groundwater, and

in treated waterways where the herbicide was used to control aquatic weeds.

In all instances, concentrations in water were low and degradation (biological rather than chemical) was rapid. 2,4-D is not a persistent water contaminant. Concentrations of 2,4-D in water will rapidly decrease as a result of dilution, microbial degradation, and photodecomposition (Bovey and Young 1980).

Bovey and Young (1980) reviewed six studies of the movement or contamination of water by phenoxy herbicides in the forest environment. Losses via runoff are a very small fraction of total amounts applied. Following spraying of forest land in Scotland with an emulsifiable formulation of the nonyl ester of 2,4-D (4.48 kg/ha), concentration in the drainage water was 2 ppm, 1, 2, 4, and 7 days after a 14 cm rainfall the day before treatment. After 28 days, no 2,4-D was detectable.

In a Great Lakes forest clearcut area of Canada, the losses of picloram and 2,4-D from the soil were measured for one year following spraying. Less than 1 percent of the picloram and less than 0.1 percent of the 2,4-D were lost during seven periods of runoff. A storm which occurred 24 hours after application failed to release significant quantities of either herbicide. Suffling et al. (1974) concluded that the major loss from soil was decomposition rather than herbicide movement.

Chemical hydrolysis is the primary mechanism of 2,4-D decomposition in water. Hydrolysis is pH and temperature dependent. Cohen et al. (1981) calculate the half-life for hydrolysis of 2,4-D esters in northern California coastal streams to range from several days to several weeks.

Norris and Moore (1970) and Norris (1970) reported herbicide concentrations of less than 0.1 ppm in streams adjacent to carefully controlled forest spray operations in Oregon. Concentrations exceeding 1 ppm have never been observed and were considered unlikely. BPA has conducted an herbicide residue monitoring program over the past 10 years. The highest reported concentration of 2,4-D in water was 0.216 ppm in a beaver pond on the day the right-of-way was aerially treated.

Air

Spray operations and volatilization may result in the occurrence of 2,4-D in air. Drift of airborne spray particles (aerosols) to nontarget plants is a significant hazard of

2,4-D spray operations. Low temperatures and high relative humidity tend to reduce volatilization. Techniques which minimize drift are important to effective and efficient spray operations.

The concentration of 2,4-D ester in ambient air during the spray season in Canada and the Pacific Northwest varies from 0.000 to 0.010 mg/m^3 (Adams et al. 1974). Bamesburger and Adams (1966) collected 24-hour air samples at Pullman and Kennewick, Washington, for approximately 100 days. Maximum concentrations of the phenoxys were the methyl esters of 2,4,5-T and 2,4-D at 0.00338 and 0.00512 mg/m^3, respectively. These studies demonstrate the transport of the phenoxys from large-scale application in agriculture. Negligible amounts would be expected in air as a result of herbicide vegetation management by BPA. Dilution is the primary process responsible for reduction of the concentration of phenoxys in air. Photodegradation also probably results in the reduction of airborne herbicide.

Chemical Toxicology in Animals and Humans

Acute Effects

2,4-D is classed as a moderately toxic substance based upon toxicity testing in an extremely large number of animals. The acute oral LD50s of 2,4-D in mice, rats, guinea pigs, rabbits, swine, sheep, cattle, and monkeys fall within the range of 300 to 1,000 mg/kg. Dogs are more susceptible with an acute oral LD50 of approximately 100 mg/kg.

Gehring and Betso (1978) reported on the low to moderate acute toxicity of phenoxy acids. They noted the greater sensitivity of the dog and associated that finding with the poor renal excretion of organic acids by this species. Signs of toxicity were: loss of appetite, weight loss, depression, muscular weakness, paresthesia (tingling of the skin), ataxia (loss of coordination), peripheral neuropathy, and posterior paralysis. Lesions are not remarkable and are limited to inflammation of the gastrointestinal tract and mild liver and kidney damage.

Acute toxicity can result from dermal exposure. The acute dermal LD50 of 2,4-D in the rabbit is greater than 10,000 mg/kg (WSSA 1979). Skin absorption of 2,4-D is limited, however. About 5 to 6 percent of dermally applied 2,4-D was recovered in human urine (Feldman and Maibach 1974). Newton and Norris (1982) recovered less than 0.5 percent of a related phenoxy ester when applied in a saturated cloth pressed against the skin for two hours. Skin exposure can result in systemic toxicity (see Hazard Assessment section),

but normally simple hygiene is sufficient to adequately minimize the hazard.

Excessively large dosages of 2,4-D may alter the physiological properties of the blood brain barrier. Desi et al. (1962) reported that 200 mg/kg altered brain electrical activity in the rat. Elo and Ylitalo (1977, 1979) administered high doses of 2,4-D followed by a trace of ^{14}C-2,4-D. At 250 mg/kg increased amounts of ^{14}C entered the brain. The breakdown in resistance to 2,4-D entry into the brain became dose-dependent only when a massive dose had been administered. Pritchard (1980) has demonstrated in vitro that the choroid plexus of the rabbit is extremely efficient (accumulating concentrations 40 to 60 times over medium) in concentrating 2,4-D (and other acids). The system is saturable. It seems likely the retention of ^{14}C-2,4-D results from saturation of the transport system with high doses of 2,4-D.

Approximately 20 cases of human poisoning by 2,4-D have been reported (Newton and Dost 1981). A single oral dose of 3 to 4 grams is necessary to produce poisoning in humans (Hayes 1963). Poisonings have resulted from spillage of herbicide concentrates on the skin without washing, sustained contact with large quantities of diluted materials, and accidental (or deliberate) ingestion. In another case, a home gardener was exposed to 2,4-D on her hands, knees, and legs for an extended period.

A unique neuromuscular effect has been observed in persons exposed to excessively large amounts of 2,4-D (Berwick 1970; Berkeley and Magee 1963; Seabury 1963; Todd 1962; Goldstein et al. 1959). In these cases, early symptoms included weakness and numbness or tingling of the extremities, followed by nausea and vomiting regardless of the route of exposure. Muscle aches which may have required medication also have followed. In come cases, patients were unable to walk. Reflex deficits were reported, and electromyography showed diminished muscular function. Although decreased nerve conduction velocity is an important symptom, the underlying mechanism of this neuromuscular disorder is not known. Clinical recovery occurred after a few months or up to two to three years.

At high dosages, 2,4-D is an experimental inducer of myotonia, used as a model of the congenital disease in humans (Iyer et al. 1977); Ezaquirre et al. 1948). The action is apparently due to increased membrane resistance and decreased chloride transport. Dux et al. (1977) concluded that 2,4-D altered calcium distribution in muscle.

In one method of interpreting toxicity data, it is presumed that acute (LD50) toxic doses for rat populations are similar to that for human populations. Given LD50 values ranging between 375 and 1,960 mg/kg for rat populations, it is presumed that the LD50 dose of various 2,4-D formulations (active ingredient) for a population of 165-pound humans would range from 1.0 to 5.2 ounces (0.006-0.032 oz/lb.).

Chronic Effects

Bovey and Young (1980) made the following summary statement: "Mice were not affected at dosages up to 93 mg/kg of the sodium salt of 2,4-D when fed daily for three weeks to three months (Bucher 1946). Rats were not affected when fed dosages up to 400 mg/kg daily for 30 days of the sodium salt of 2,4-D (Hill and Carlisle 1947). A dosage of 500 mg/kg for 30 days, however, was lethal. Hansen et al. (1971) and Erne (1966) fed the acid and amine formulation of 2,4-D to rats up to 1,000 ppm daily for two years and ten months, respectively, with no detrimental effects". These studies indicate the low order of chronic toxicity of 2,4-D.

The report of Hansen et al. (1971) is most extensive and provides the most unified data on 2,4-D chronic toxicity in rats and dogs. A two-year chronic feeding was done using Osborne-Mendel rats (25 male, 25 female) fed 0, 5, 25, 125, 625, or 1,250 ppm 2,4-D. No significant effects on growth rate, survival rate, organ weights, or hematologic values were noted. Total tumor incidence (male and female in control and experimental groups), and tissue distribution of the tumors was typical of aging rats. 2,4-D was not carcinogenic.

A two-year chronic feeding study was also performed in beagle dogs. They (3 male, 3 female) were fed either 0, 10, 50, 100, or 500 ppm 2,4-D. Necropsy was performed on a male (on 10 ppm) which died after ten months, and all of the 29 others, which survived for two years. Tissues were studied grossly and microscopically. Twenty-eight dogs which survived two years were clinically normal, and no 2,4-D effects were observed. One female at 100 ppm was emaciated at autopsy but showed no significant lesions . None of the lesions in the dogs was believed to be due to ingestion of 2,4-D.

The effect of 2,4-D on reproductive performance by rats was studied with a total of six litters produced by three successive generations of rats. Dose levels of 2,4-D were 100, 500, and 1,500 ppm. At the highest dose, there was no effect on fertility of the male or female rats or on average

litter size, but 1,500 ppm did reduce the percent of pups born that survived to 21 days and depressed the weights of those weanlings.

The long-term study of Innes et al. (1969) also failed to show any carcinogenic activity of 2,4-D in two strains of mice.

Hansen et al. (1971) calculated the maximum allowable human 2,4-D exposure based upon registered tolerances in fruits (5 ppm) and grains (0.5 ppm). They estimated 2,4-D could comprise 0.3 ppm of the total diet.

Studies of the teratogenic activity of 2,4-D have sometimes produced serious abnormalities (terata), but the threshold dose is very close to those that cause maternal toxicity. Schwetz et al. (1971) fed 2,4-D acid at levels up to 87.5 mg/kg/day on days 6 to 15 of gestaton. Embryotoxicity (edema, delayed ossification, decreased fetal weight, and wavy ribs) was apparent, but no terata were seen. Khera and McKinley (1972) produced skeletal anomalies after single oral doses of up to 500 mg/kg/day on days 6 to 15 of gestation. The lowest dosage at which increased incidence was observed was 25 mg/kg/day. Collins and Williams (1971) used three commercial samples of 2,4-D at levels up to 100 mg/kg/day in hamsters on days 6 to 10 of gestation. The incidence of fused ribs was elevated but not significantly above control values.

As do many chemicals, 2,4-D has the capacity to cause subtle reproductive effects (e.g. decreased birth weights, decreased litter size, decreased fertility). Most such effects occur at doses in the range where adult toxicity is caused. The dose at which no adverse changes in reproduction have occurred has been determined at 25-35 mg/kg/day. On this basis, Dost (1983) used 20 mg/kg/day as the reproductive no-effect level for 2,4-D in a worst-case analysis of environmental herbicide exposure.

A number of mutagenic screens have also included 2,4-D. Negative, weakly positive, and positive results have been reported and are summarized by Newton and Dost (1981). In a screen of 110 herbicides using eight strains of histidine-requiring mutants of bacteria (Salmonella typhimurium), 2,4-D failed to produce point mutations (Anderson et al. 1972). Styles (1973) treated rats with 2,4-D. The serum of the treated rats was used in a host-mediated assay with histidine-requiring S. typhimurium mutants. 2,4-D did not induce increased micronuclei in mouse bone marrow erythrocyte, but mitotic activity was slightly depressed (Jenssen and Renberg 1976). The sex-linked lethality assay of 2,4-D

in male fruit flies (Drosophila) was negative (Vogel and Chandler 1974), weakly positive (Magnusson et al. 1977), and positive (Rasmussen and Svahlin 1978) in three separate studies.

Seiler (1978) reviewed the genetic toxicology of phenoxy acids including 2,4-D. He concluded that any genetic risk associated with these herbicides is apparently low. Newton and Dost (1981) concluded that 2,4-D may be a very weak mutagen, "but is without significance as an environmental mutagenic hazard."

Some inhibitors of plant cell division are capable of inhibiting the proliferation of animal tumor cells. Two animal tumor cell cultures, EL-4 and L1210 were used. Zilkah et al. (1981) found that tumor cell proliferation was inhibited 50 percent by concentrations of 10^{-4} to 10^{-3}M of 2,4-D and bromacil.

Mode of Action

Factors which determine how much chemical interacts with a sensitive site in the body include absorption, distribution, biotransformation, storage, and excretion. Knowledge of these processes is important to predicting the outcome of accidental or unavoidable exposures.

2,4-D is readily absorbed from the gastrointestinal tract. In human volunteers, blood levels reached 80 percent of maximum concentrations two hours after a 5 mg/kg dose (Kohli et al. 1974; Sauerhoff et al. 1977). Absorption occurs via the portal vein rather than through the lymphatic system as a result of the low fat solubility of 2,4-D (Sieber 1976).

In mammals, there is relatively little conversion of 2,4-D acid to other metabolic products. The various salts and esters are readily converted to the parent acid. In humans, Sauerhoff et al (1977) found 82 percent of the 5 mg/kg dose as unchanged 2,4-D in urine and 12.5 percent as a conjugate (probably combined with the amino acids taurine and glycine) (Grunow and Bohme 1974). In the studies of Kohli et al. (1974) only 2,4-D was recovered.

Excretion of 2,4-D is very rapid. Kohli et al. (1974) found an average of 33 hours was required for elimination of 2,4-D.

Sauerhoff et al. (1977) found an average half-life of 18 hours (range 10.2 to 28.5 hours). Feldman and Maibach (1974) reported a 2,4-D half-life of 13 hours following dermal exposure.

2,4-D is readily distributed to all tissues and then is lost readily without biotransformation. The excretion mechanism is saturable so large doses are eliminated more slowly than small doses (Khanna and Fang 1966; Erne and Sperber 1974).

Confirmation of the rapid elimination of 2,4-D was obtained by measurements of urinary 2,4-D following a suicidal exposure of approximately 250 mg/kg. The 2,4-D half-life was 17 hours, well within the range established in the previously mentioned studies with human volunteers.

Potential Impact on Nontarget Organisms

Vegetation

Drift of aerosols and volatilization are means of nontarget plant exposure to 2,4-D. Most established grasses are not harmed by 2,4-D; however, plants such as cotton, grapes, tobacco, beans, tomatoes, fruit trees, and some ornamentals are extremely sensitive. Conifers are sensitive to 2,4-D during periods of rapid growth.

Fish and Other Aquatic Organisms

Since 2,4-D has been extensively used to control aquatic weeds such as water hyacinth and water milfoil, it has been studied under laboratory and field conditions. References to studies in fish and other aquatic organisms number in the hundreds.

The various 2,4-D formulations vary considerably in their acute toxicity (Pimentel 1971; Bovey and Young 1980). The ester formulations are the most toxic due to their more extensive uptake by aquatic organisms (Hughes and Davis 1963). For example, the 24-hour LC50s of the sodium salt and the butyl ester of 2,4-D are 1,160 and 1 ppm, respectively (Pimentel 1971).

The LC50s in rainbow trout of 2,4-D amine and propylene glycol butyl ether ester are 250 ppm (24-hour) and about 1 ppm (48-hour) (Pimentel 1971).

Folmar (1976) reported a 96-hour LC50 of 100 ppm for rainbow trout exposed to 2,4-D amine. The trout avoided concentrations of 1 ppm but not 0.1 ppm.

Young silver salmon when exposed to 2,4-D and 2,4,5-T at concentrations of 50 ppm or more were "immediately distressed and would snap their jaws, dart about the aquarium,

and leap out of the water before loss of equilibrium and death (Holland et al 1960)."

The 96-hour LC50 for 2,4-D (amine) in yearling coho salmon was greater than 200 ppm. 2,4-D did not affect the downstream migration of smolts nor their survival in seawater (Lorz et al. 1979).

Woodward and Mayer (1978) studied the acute and chronic toxicity of three 2,4-D esters on cutthroat and lake trout. 2,4-D butyl ester was slightly more toxic (96-hour LC50, 490 and 600 ppb [microgram per liter]) than 2,4-D propylene glycol butyl ether ester. Susceptibility increased as temperature decreased. Neither water hardness nor pH influenced sensitivity. The 2,4-D isooctyl ester was not toxic to either species at 60,000 ppb (=60 ppm). No effect concentrations of the butyl ester and the propylene glycol butyl ether ester were 24 ppb and 33 ppb, respectively, for cutthroat trout. The corresponding concentrations were 31 ppb and 52 ppb for lake trout. The ratios of 96-hour LC50 to the no-effect concentrations were 10 to 20, indicating a relatively low chronic toxicity hazard.

The acute toxicity of 2,4-D acid, butyl, and isooctyl esters to juvenile salmon, char, and rainbow trout was measured (Meehan et al. 1974). Concentrations of acid less than 50 ppm produced mortality only in pink salmon fry. The butyl ester was the most toxic, causing nearly complete mortality in all species at concentrations greater than 1 ppm.

Norris and Moore (1970) and Norris (1970) found that when buffer zones and other application guidelines were observed, concentrations of 2,4-D in streams never exceeded 1 ppm. In the cases of the more toxic 2,4-D esters where LC50s between 100 ppb and 10 ppm are common (Newton and Dost 1981), the initial concentrations in water might be in the same or within one order of magnitude as the acute LC50 for some fish, especially larvae or eggs. Hydrolysis of the ester to a less toxic form usually occurs in water, but is pH and temperature dependent (see Fate in Water section). The use of 2,4-D esters in vegetation management may affect fish. The use of other 2,4-D compounds in vegetation management is not likely to affect fish because of the significantly higher LC50 concentrations.

The effective concentrations for other aquatic organisms also vary with the form of 2,4-D. The 24-hour LC50 for chorus frog tadpoles was 100 ppm 2,4-D (Pimentel 1971). Other studies with amphibians are listed by Bovey and Young (1980). Exposure of eastern oysters to 2 ppm 2,4-D for 96 hours had no effect on shell growth (Pimentel 1971). Field

applications at rates of up to 120 pounds/acre did not affect oysters and clams.

Cohen et al. (1981) have reviewed data on the toxicity of the propylene glycol butyl ether ester of 2,4-D to various aquatic arthropods. Daphnia (water fleas) are the most sensitive, having an acute LC50 (48 hrs.) of 0.062 ppm. Ostracods (seed shrimp) are also very intolerant, having an acute LC50 (48 hrs.) of 0.197 ppm. Amphipods, isopods, and stoneflies displayed LC50s ranging from 1.3-1.6 ppm. These results indicate the potential for chronic toxicity, and potentially acute toxicity of this ester derivative of 2,4-D.

Sublethal effects on invertebrates are poorly understood. The available information suggests that indirect impacts may result from sublethal concentrations, especially of the more toxic ester derivatives, but the cause-and-effect relationships would be difficult to establish (Bowes et al. 1981).

Wildlife

The acute oral LD50 of 2,4-D has been assessed in some game birds and mule deer. Data tabulated by Tucker and Crabtree (1970) indicated that the LD50 to young mallards was much greater than 1,000 (acid) mg/kg and much greater than 2,025 (sodium salt) mg/kg. The LD50s for young pheasants, Japanese quail, and pigeons were 472, 668 and 668 mg/kg, respectively. Feeding studies using two-week-old mallards, pheasants, Japanese quail, and bobwhite quail showed that the dietary LC50 was greater than 5,000 ppm when fed treated feed five days, followed by untreated feed for three days (Pimentel 1971). The phenoxy herbicides have a low order of acute oral toxicity in birds as indicated by the above findings.

Sublethal dietary exposures (500 to 5,000 ppm) had marked inhibitory effects on bird reproduction (DeWitt et al. 1963, cited in Bovey and Young 1980). Effects were observed at dosages of 10 to 50 percent of estimated lethal dosages. Details of the inhibitory effects were not reported.

Canada geese fed diets contained ^{14}C-2,4-D were killed after 22, 50, 62, 73, 113, 134, 197, and 230 days after treatment. At necropsy, enlarged kidneys and jaundice of other organs were observed. Weight gain was depressed. Tissue necrosis was noted in kidney and liver. The effects were reversed seven months after cessation of the 2,4-D feeding (Sheldon et al. 1963).

Dimethylamine salt of 2,4-D was sprayed on eggs of quail and gray partridge at 1.12, 2.4, and 6 kg/ha. Rate of hatching,

embryonal and postnatal mortality, and teratogenic malformations were examined in hatched and unhatched embryos. No harmful effects on reproduction were reported. 2,4-D residues in eggs were low due to limited penetration through the egg shell (Grolleau et al. 1974).

A feeding study in reindeer was prompted by allegations that a high incidence of death and abortion occurred in 1970 in a herd after use of phenoxy herbicides the previous year. Fifteen of 30 pregnant reindeer were fed for six weeks late in gestation birch leaves that had been sprayed with 2,4-D and 2,4,5-T. The daily dosage was about 1 mg/kg/day. No clinical changes were detected and no changes were evident at necropsy of adults and full-term fetuses (Erne 1974).

Livestock

Cattle and sheep grazing on 2,4-D treated pasture and a cow fed 5.5 g of 2,4-D daily in the ration for 106 days demonstrated no harmful effects (Mitchell et al. 1946). 2,4-D was detected in serum but not in milk, liver, kidney, or fatty tissue. A plant bioassay was used in this early work. Later studies utilized sensitive gas chromatographic analysis. Pasture was sprayed at a rate of 2.24 kg/ha with isooctyl and isopropyl esters. Mean 2,4-D levels in milk were 0.03 ppm after 12, 24, and 36 hours. No residues were detected after four days, indicating rapid removal of 2,4-D from cattle (Boyce Thompson Institute for Plant Res. 1962).

Gutenmann et al. (1963) found no 2,4-D in milk or feces from a cow fed 5 ppm in diet for five days. These workers also demonstrated the conversion of 2,4-DB to 2,4-D (Bache et al. 1964).

In field trials, Klingman et al. (1966) found from 0.01 to 0.09 ppm 2,4-D in milk during the first two days after spraying and less thereafter. If cows were put into the sprayed pastures four days after 2,4-D application, no residues were found in milk.

Sheep excreted 2,4-D unchanged after an oral dose of 4 mg/kg. Approximately 96 percent of the 2,4-D was recovered in urine within 72 hours. Edible tissue contained less than 0.05 ppm 2,4-D (Clark et al. 1964).

Beef calves fed 28 days at 2,4-D dietary levels of 100, 1,000, and 2,000 ppm did not have detectable 2,4-D in muscle (limit of detectability 0.05 ppm) (Miller and Gentry 1970).

Since 2,4-D is not accumulated in sheep and cattle, they may be placed on 2,4-D-free feeds for one week to assure negligible transfer of residue to consumers (Bovey and Young 1980).

No adverse effects on incubation and subsequent performance of hatched chicks resulted from aqueous sprays of 2,4-D: picloram (4:1) at recommended and up to 20 times field rates. The amounts of 2,4-D in the shell, interior shell, and the chick were 26.6, 0.49, and 0.31 ppm, respectively (Somers et al. 1974).

Hazard Assessment for Current Use Practices

2,4-D is a phenoxy herbicide. Based upon that chemical classification and its widespread use as an herbicide, 2,4-D has been the focus of much public controversy. Reconsideration of some of the information presented in the first section of this Background Statement is warranted at this time. Both 2,4-D and 2,4,5-T are derivatives of phenoxyacetic acid. 2,4-D is the dichloro derivative and it does not contain tetrachlorodibenzodioxin (TCDD) contaminants. The herbicide 2,4-D in its acid, ester, and amine formulations is one of the most versatile agents available for the control of broadleaf weeds. The amine formulation is also utilized as a stem frill, notch, and injection treatment against unwanted trees.

Under field and laboratory conditions, 2,4-D is among the most thoroughly tested chemicals used in pest control. It is an exceptionally effective herbicide which is readily degraded in the environment to nontoxic products. Like most herbicides, 2,4-D has a low order of toxicity and is rapidly eliminated (usually in urine) in animals. Some aquatic species are very sensitive to 2,4-D esters under experimental conditions. Concentrations exceeding 1 ppm have never been observed following use by BPA, and initial concentrations decline rapidly according to BPA monitoring studies (Norris 1971-1976, 1979).

The issues of carcinogenicity, teratogenicity, and worker safety related to 2,4-D use have come before the public and the scientific community. The results of these inquiries will be summarized in the following paragraphs.

The best sources of information about human exposures to pesticides in general, and to 2,4-D in particular, are epidemiological studies of applicators. Although such studies are extremely difficult to conduct, they provide invaluable information about the extent of human exposure and critical data on associated effects in humans.

Carcinogenicity

Axelson and Sundell (1974) reported that an epidemiological investigation of tumor incidence and mortality among Swedish railroad workers exposed to different herbicides showed a two-fold excess of cancers as compared to the national average. The situation is difficult to evaluate because of the complexity of the herbicide exposures. The excess cancer may be due to exposure to 3-amino-1,2,4-triazole (amitrol). Subgroups that had been exposed to 2,4-D and/or 2,4,5-T had about normal tumor incidence.

The International Agency for Research on Cancer has reviewed and evaluated studies (including Axelson and Sundell 1974) on the carcinogenicity of 2,4-D (IARC 1977). IARC is supported in part by the National Cancer Institute and is a source of independent, expert opinion. IARC concluded that existing data were inadequate to evaluate the carcinogenicity of 2,4-D in animals and in man.

Reproductive Effects

A case control study of the relationship between exposure to 2,4-D and spontaneous abortions (miscarriages) in humans was recently conducted (Carmelli et al. 1981). The study utilized two groups of people, "cases" and "controls", from the farmlands and forests of Washington and Oregon where 2,4-D is used extensively. Only occupational exposures were considered. The investigators used women who had experienced a miscarriage in the previous two years as their "cases". The "controls" were women of similar age and socioeconomic circumstance who had experienced full-term delivery during the same two-year period. Their exposues to 2,4-D were compared. It was concluded that: "the results of the study do not indicate any evident relationship between the use of 2,4-D and spontaneous abortion".

A finding deserving further study was a "suggestive association" (low confidence level) between paternal exposures and reproductive problems in an isolated subgroup of wives of young forest/commercial workers and overall 2,4-D exposure. It was concluded that the observation "does not in itself argue for restrictions on 2,4-D use pending such a study".

Worker Exposure

Two epidemiological studies (Kolmodin-Hedman and Erne 1980; Lavy et al. 1982) have measured occupational 2,4-D exposure in men working in ground-based and aerial application

operations. The simple pattern of metabolism and rapid excretion of 2,4-D in urine (Sauerhoff et al. 1977) are important to the success of these studies. A third study has been completed (Nash et al. 1982).

Occupational exposure to phenoxy acids (2,4-D and 2,4,5-T) has been studied in four men spraying 2 percent emulsion in kerosene from tractor-driven equipment (Kolmodin-Hedman and Erne 1980). Airborne concentrations in the breathing zone were 0.1 to 0.2 mg/m^3. The highest urine levels were found the afternoon of a day of exposure (mean 2,4-D was 0.008 mg/ml; range 0.003 to 0.014 mg/ml). The mean 24-hour urinary excretion of 2,4-D was 9 mg. Dermal and inhalation absorption occurred. Exposure could be reduced by improved hygiene. No adverse health effects were observed. No 2,4-D or 2,4,5-T could be detected in plasma and urine of control subjects with low and indirect exposure.

The 2,4-D exposure received by aerial application crews during forest spray operations was measured by Lavy et al. (1982). Levels of 2,4-D were measured in air near the breathing zone, on denim patches to estimate dermal exposure, and in urine (two days before and five days after the spraying). Each crew made two applications about one week apart to compare exposure of crew members wearing customary clothing and following normal precautions (T-1) to the exposure of workers wearing protective apparel and following special hygienic practices (T-2).

Exposures were reported in terms of mg/kg for individual members of the work crews (helicopter pilot, mechanic, batchman loader, supervisor) plus two observers. Air samples did not contain 2,4-D (0.05 microgram limit of detectability). Based upon the amount of 2,4-D recovered from the denim patches, the external exposures ranged from nil to 0.0911 mg/kg (Batchman in T-1).

The total worker exposures were estimated by summing the amounts recovered in the seven-day urine collections. Less than 30 percent of the 524 samples analyzed contained detectable 2,4-D (0.04 ppm limit of detectability). Exposures in T-1 ranged between nondetectable and 0.0557 mg/kg. The highest exposure in T-2 was 0.0237 mg/kg. Crewmen who worked most closely with spray concentrate or who handled spray equipment had the highest exposures. Protective clothing and good hygienic practices limited exposure (Lavy et al. 1982).

Conclusions

The results of these inquiries concerning carcinogenicity, reproductive effects, and worker exposures supplement an extensive literature on the toxicology and environmental fate of 2,4-D. This herbicide stands as one of the most thoroughly studied chemicals used as a pesticide. The use of 2,4-D in accordance with label instructions and under conditions prescribed by the Transmission Line Maintenance Standard results in a low degree of hazard to personnel, the public, and the environment. Potential hazards to aquatic environments (especially due to 2,4-D esters) are minimal if maintenance standard constraints (e.g., stream buffer zones) are strictly observed.

Addendum

Much previous testing on the toxicity of 2,4-D (e.g. all three previous laboratory studies of carcinogenicity) was conducted using methodologies that are outdated by present EPA standards. As a result, an industrial task force of 12 manufacturers of 2,4-D is conducting additional chronic testing. "Data gaps" (i.e. lack of information) rather than evidence that adverse health effects are possible have triggered this additional testing.

The additional studies include several types of chronic toxicity evaluations. A two-year rat feeding study is expected to provide results in 1986. Range-finding studies in mice (90-day) have been completed and two-year carcinogenicity studies are also under way. Neurotoxicity studies and teratology studies are both scheduled to be completed in 1983. The results of these studies will be filed with EPA as part of the continuing herbicide registration process.

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DIESEL OIL

Chemical Identification

Diesel oil is the common name for a group of petroleum products which are also referred to as diesel fuel, No. 2 fuel oil, No. 2 heating oil, and gas oil. The composition and the appropriate marketing name of diesel oil depends on the nature of the original crude oil and the processing at the refinery, including additives which may be used for specific purposes. BPA uses diesel No. 2 as a carrier for five of the herbicides used in its vegetation management program. Diesel oil is a complex mixture of hydrocarbons having a boiling range of about 350-700°F. The characteristics of diesel oil and the terms used to describe groups of hydrocarbons are more easily understood after briefly reviewing the refinery process. This is germane from a toxicological point of view, because some hydrocarbons are more toxic or contaminating to plants and animals than others.

Crude oil is a complex composite of hydrocarbon molecules which can be subdivided into three main classes: (1) alkanes (paraffins), which are saturated chain compounds, either straight-chained (n-alkanes or normal alkanes) or branch-chained (isoalkanes or isoparaffins); (2) cycloalkanes (naphthenes), which are saturated cyclic compounds; and (3) aromatics, which are based on the benzene ring, a nonsaturated ring compound. During refining, crude oil is first separated into "cuts" of different boiling ranges. Longer molecules boil off at higher temperatures, therefore, each cut is composed of compounds with similar numbers of atoms (i.e., similar lengths). The basic cuts and approximate number of carbon atoms are: (1) refinery gases (C_3-C_4); (2) gasoline (C_4-C_{10}); (3) naptha (C_{10}-C_{12}); (4) kerosene (C_{12}-C_{16}); (5) gas oil (C_{16}- C_{25}); and (6) residual oil (above C_{25}). Basic cuts may be further refined and mixed to provide marketable products. Diesel No. 2 contains predominately n-alkanes ranging from C_{10}-C_{22}. Diesel No. 2 also contains naphthenes, alkenes (unsaturated straight-chained compounds such as olefins) produced by the refining process, aromatics which occurred in the crude oil, and additional aromatics formed during the refining process. The alkenes and aromatics are of greatest concern as potential toxicants.

The flash point of Diesel No. 2 is about 185°F (85°C). The vapor pressure varies significantly with each of the

compounds in the mixture. Low carbon (short, low molecular weight) molecules are more volatile than longer, high-carbon molecules. As a mixture, Diesel No. 2 has a vapor pressure of 2.07 mm Hg at 40°C. The specific gravity of Diesel No. 2 is about 0.82. Although diesel oil generally is not miscible with water, certain alcohols, aromatics, and phenols (a special kind of aromatic) contained in diesel oil are water soluble.

Action in Vegetation

Diesel oil readily wets plant surfaces and spreads as a thin film over leaf surfaces. Diesel oil penetrates the crowns of grasses where growth originates. It penetrates many other plants through stomata, because oil has a very low surface tension and is not barred from penetration as are most aqueous solutions (Van Overbeek and Blondeau 1954). Diesel oil, therefore, may increase the absorption by the plant of systemic herbicides.

Numerous workers suggest that an oil coat on leaves and stems will inhibit gas exchange and lead to death, but the data do not support this suggestion (Baker 1970). Kerosene and diesel fuels are contact herbicides in their own right (California Department of Food and Agriculture 1978), because of the toxic properties of the more volatile components.

Oil moves into intercellular spaces, and in the process may interfere with translocation of metabolic products and nutrients in the plant (Baker 1970). Oil does not penetrate cells until the cells are injured (Van Overbeek and Blondeau 1954). Toxicity varies according to the content of low boiling compounds, alkenes (unsaturated compounds), aromatic compounds, and acids. Phenolic acids and polycyclic aromatics are especially toxic to higher plants at low concentrations because of disruptive effects on cell membranes (Van Overbeek and Blondeau 1954; Larson et al. 1977). Chronic injury also results from alkenes (Baker 1970).

The mechanism of cell injury and death is not clearly understood. Oil application depresses phytosynthetic rates and depresses respiration rates to a lesser extent. As a result, respiration may exceed photosynthesis and lead to cell death (Wedding et al. 1952). Van Overbeek and Blondeau (1954)have described the symptoms of oil toxicity. The earliest symptom is darkening of young leaf tips, presumably because of fluid leakage into intercellular spaces. The darkening spreads to older leaves, and cells begin to lose turgor, resulting in plant drooping. Eventually cell membranes are disrupted, resulting in an increase in cell

membrane permeability. Photosynthetic activity drops after chlorophyll is destroyed by bright sunlight. Photosynthesis is inhibited by oil deposition as low as 0.3 to 0.6 mg/cm^2 (leaf surface).

Although aged crude oil is generally characterized by a reduction in the toxic components, exposed fuel oil contains a number of newly-formed oxidized compounds which are toxic, including reactive peroxides and acids. Early acute phototoxicity is due almost entirely to reactive peroxides (Larson et al. 1977). In certain respects, aged fuel oil may be more dangerous to plants than fresh fuel oil because chronic injury to cell membranes is most likely to occur in the presence of peroxides, nonvolatile acids, and oxidized aromatics (Baker 1970; Larson et al. 1977) formed in the presence of light and air (Young and Sethi 1975).

Utilization by BPA

Diesel No. 2 fuel oil is used as a carrier by BPA during directed stem or stump applications of Banvel 4-O.S. (dicamba) and Weedone 170 (2,4-DP ester and 2,4-D ester). It is not used during aerial or ground foliage application of herbicides. Only Banvel 4-O.S. is mixed solely with oil.

Banvel 4-O.S. is applied with portable sprayers to basal stems of target vegetation. The application is made until runoff occurs on the basal part of the stem. Weedone 170 may be mixed in oil or in an oil and water mixture. In these media, it is applied to the base of stems or to cut surfaces. Cut surfaces or the basal part of stems are thoroughly drench.

Chemical Fate and Distribution in the Environment

A great body of knowledge exists on the fate of oil, especially crude oil, in the marine and estuarine environment. Very few studies have been conducted on the fate of diesel fuels in terrestrial or freshwater environments.

Soils

Aging or weathering of oil is often considered to reduce the quantity of spilled oil because of the rapid evaporation of

volatile (low-boiling point) hydrocarbons. Diesel fuel contains proportionally few low-boiling hydrocarbons; therefore the losses from soil depend primarily on microbiodegradation and leaching.

Aliphatic (nonaromatic) hydrocarbons are usually adsorbed to soils, and slowly evaporate or undergo biological degradation; they do not leach readily. Soluble aromatics such as alkylated benzenes and naphthalenes tend to be volatile, but also tend to remain stable and are mobile in groundwater (Zurcher and Thuerr 1978). Bertsch et al. (1975) found that water soluble oil components move readily with groundwater movement. Thofern (1962) did not detect diesel oil (detection level of 1 ppm) in a stream adjacent to a spruce plantation treated with 15-20 gallon/acre (14-19 ml/m^2) of diesel oil, despite a heavy rainfall immediately after application.

Water

Diesel fuel first forms a partial film on surface water. Although the acutely toxic, volatile compounds may quickly evaporate from the film, these same compounds tend to be soluble in water. Photooxidation acting on surface films can generate materials highly toxic to aquatic organisms. Crude oils appear to be less susceptible to photooxidation because sulfur compounds (e.g., heterocyclic sulfides), which effectively inhibit radical reactions, have not been refined out (Larson et al. 1977). Surface oil is readily adsorbed on suspended particulates, and may sink to the stream bed. Once in the sediments, n-alkanes are most readily degraded, whereas cyclic, branched, and aromatic compounds are most stable and resist microbial degradation (Wakeham and Carpenter 1976). Blumer (1970) found that No. 2 fuel oil incorporated into estuarine sediments ater a spill persisted for over a year, and spread in the form of oil-laden sediment beyond the original spill area.

Oil particles in water are decomposed mainly by aerobic microbiological processes, but the process requires oxygen (McCauley 1966). The BOD is about 3.1 to 3.5 mg O_2/mg oil, or about 2,500 to 2,800 mg O_2/ml oil (assuming a density of diesel fuel = 0.8/ml). Water temperature is important in determining the rates of decomposition and sedimentation.

Air

Low molecular weight constituents quickly volatilize and enter the atmosphere. Aromatic compounds and alkanes (C_4 and higher) are stable and fairly resistant to photochemical degradation. Alkenes readily undergo photochemical degradation in ultraviolet light, and react with oxygen, ozone and oxides of nitrogen to form smog.

Chemical Toxicology in Animals and Humans

There are few toxicological data available on the ingestion of oil by test animals or humans. Most data deal with impacts on fish and wildlife following an accidental spill, or result from laboratory tests on fish and wildlife. Studies of isolated petroleum constituents are of little value because of the difficulties of applying the data to a complex mixture such as diesel No. 2.

Bruns et al. (1955) found that guinea pigs display an aversion to drinking water treated with 400 and 800 mg/l (ppm) of aromatic hydrocarbons (mostly xylene). The guinea pigs refused the higher concentration until the third day. None of the animals showed ill effects after three days of confinement on treated water.

The acute oral LD50 of kerosene for rabbits is 28,350 mg/kg body weight (Spector 1955).

Potential Impact on Nontarget Organisms

Vegetation

Nontarget vegetation which is most likely to be of concern to BPA is aquatic vegetation and crops irrigated by water from treated watersheds.

Aromatic constituents of diesel fuels are known to be toxic to aquatic plants. Aromatic solvents have been used at 10,000 ppm to kill vegetation rapidly in irrigation ditches (Currier and Peoples, 1954 cited in Baker 1970).

Oil films on the surface do not prevent growth of aquatic plants (Roberts 1930); the hazard is due to soluble constituents. Perennial freshwater marsh plants are generally less affected by fuel oil than annual species immediately following a spill (Burk 1977).

Straus (1949) reports that a concentration of 300 ppm in the stream for 30 minutes will kill aquatic weeds without

harming crops subsequently irrigated. The loss of toxicity in 30 minutes probably occurred because of volatilization and dilution.

Light oil sprays have been used for many years to control mites on citrus crops (Wedding et al. 1952). These oils have been of minimal hazard to bees on blooming crops (California Department of Food and Agriculture, 1978).

Fish and Aquatic Organisms

Oils of all kinds may impact fish because of toxic constituents, lethal effects of oil coating gill epithelial surfaces, elevated BOD and impacts on food organisms. Among oil components, saturated aliphatic compounds are not lethal to fish, but monocyclic aromatic components were generally toxic, and the degree of toxicity increases with the degree of unsaturation (Morrow 1974).

The 24-hour LC50 for American shad exposed to diesel fuel is 204 ppm (Tagatz 1961). U.S. EPA (1976) reports a 96-hour LC50 greater than 1.20 ppm as the acute toxicity of No. 2 fuel oil to freshwater fish, and greater than 0.19 ppm as the acute toxicity of diesel fuel to freshwater fish, assuming a density of 0.8 g/ml. The lethal limit of gasoline for rainbow trout and fingerling salmon is about 0.13 ppm (McKee and Wolf 1963).

Benzene at 5 ppm and 10 ppm causes an initial increase in the respiration of chinook salmon (U.S. EPA 1976).

Exposure to ultraviolet light increases the toxicity of the water-soluble fraction of No. 2 fuel oil (Scheier and Gominger 1976). Morrow (1974) found that crude oil exposed to air for 30 days produced no significant mortalities on young salmon, but fresh crude oil produced significant mortalities at concentrations of 500 ppm or greater. Weathering of crude oil probably does not increase toxicity because of the presence of sulfonated compounds, which are not found in refined petroleum. Weathering of diesel fuel does increase toxicity, and may account for differences in reported acute toxic levels.

Phenolic compounds cause tainting of fish flesh, e.g., o-chlorophenol produced an off-flavor in bluegills at 2 ppm (Pickering and Henderson 1966). Rainbow trout exposed 24 hours to 10 mg/l No. 2 fuel oil had an off-flavor (Kopperdahl et al 1975). Exposure to 5 mg/l for 5 days also imparted an off-flavor.

Oils are toxic to mosquito larvae, and to numerous aquatic invertebrate species. Crayfish have been eliminated from irrigation ditches when concentrations of 300 mg emulsified aromatic hydrocarbons/l were used to destroy aquatic weeds (McKee and Wolf 1963). Phytoplankton species vary significantly in their response to oil contamination (McCauley 1966).

Wildlife

The acute oral LD50 dosage of diesel oil for mallard ducks over one year old is greater than 20 ml/kg (16,000 mg/kg, assuming density is 0.8 g/ml) (Tucker and Crabtree 1970). Birds may ingest diesel oil either by eating contaminated foods or during preening. Hartung (1965) reported that a duck ingests about one-third of the oil sprayed on its feathers.

Birds, especially waterfowl, may be impacted in several ways other than ingestion of oil. Oiling reduces buoyancy of waterfowl, potentially leading to drowning. Oiling also reduces the insulating capacity of feathers, leading to death from exposure. Starvation or death from predators also may occur because of a loss of flight capability. These effects are not likely to occur from BPA application of diesel oil unless birds, bird nests, or water surfaces are accidentally sprayed.

Although mallard ducks can ingest a relatively large dose of oil without ill effects, traces of oil in the diet sharply reduce egg production (Biderman and Drury 1980). Application of oil to incubating eggs substantially impacts hatchability. Hartung (1965) found a direct relationship between the degree of oiling and the hatchability of mallard eggs. More than 10 mg oil/egg virtually eliminated hatching success. Szaro et al. (1978) noted that application of micrograms per liter (about 4 mg) of No. 2 fuel oil reduced hatching success to 18 percent. The data suggest embryonic death resulted from toxicity of the aromatic components, and not because of blocked gas exchange. The amount of oil on the plumage of oiled ducks can be as high as 10.6 ml (8,480 mg). Incubating birds turn their eggs regularly, therefore thorough oiling is likely.

Kopischke (1972) sprayed 57 viable pheasant eggs with diesel oil to runoff; none hatched. The same number sprayed with 2,4-D (field concentration) to runoff showed no measurable impact on hatchability.

Naphthalene, an aromatic hydrocarbon, is readily accumulated by ducks fed contaminated crayfish (Biderman and Drury 1980).

The amount of spray deposited on forage plants has been examined (Norris et al. 1975, cited in U.S. Forest Service 1979). At an application rate of 93 ml/m^2 (about 79,050 mg/m^2), forage would contain about 66,500 mg diesel No. 2 per kg of forage. If one assumes an herbivore consumes forage at a level of 10 percent of its body weight per day, ingestion would approximate 6,650 mg diesel per kg body weight, which is below the acute LD50 of kerosene of 28,350 mg/kg for rabbits (Spector 1955), and a sublethal dose of diesel oil of 16,000 mg/kg force-fed to rabbits (U.S. Forest Service 1979).

Livestock

Information on toxic effects of diesel fuel on livestock is limited. The following information is taken from Clark and Clark (1965). The toxic dose of diesel No. 2 is not available, but 12 ml (9,840 mg) of tractor oil/kg was tolerated by cows, whereas 20 ml/kg (16,400 mg/kg) was fatal to cows in 32 days. Symptoms in cattle are loss of appetite, constipation, and muscular weakness. Vomiting and rapid, shallow breathing may occur, as may acetonemia. Poisoning from kerosene has been reported after external exposure to kerosene and water applied to control lice. Symptoms may include loss of appetite, partial paralysis, severe dermatitis, and hematuria (urine in the blood).

Hazard Assessment for Current Use Practices

Diesel No. 2 is an effective carrier which dilutes herbicides insoluble in water and which increases the absorption of systemic herbicides by plants.

Based on the acute LD50 of kerosene in rabbits (28,350 mg/kg), diesel fuel, which contains proportionately fewer of the toxic, volatile hydrocarbons, is likely to be of little hazard. "Any substance with an LD50 rating exceeding 15,000 mg/kg is rated as reltively harmless". (U.S. Forest Service 1979). Diesel fuel force-fed to rabbits at 16,000 mg/kg proved to be sublethal.

The environmental fate of diesel No. 2 incicates that the greatest hazard to plants and aquatic animals will occur after the oil has weathered. The potential for diesel No. 2 contaminating nontarget plants or surface water is likely to be low, however, because of the current methods of application. The amount of photochemical smog occurring from BPA's vegetation management program probably is not measurable.

Large herbivores, including deer and livestock, are unlikely to be sprayed by diesel fuel or consume contaminated water or forage. Tolerable health concentrations far exceed the limits of taste and odor. Petroleum products become organoleptically objectionable at very low concentrations, and large herbivores will forage and drink in uncontaminated areas until oil residues are no longer detectable.

The major hazards associated with diesel No. 2 as an herbicide carrier result from impacts on birds and on small herbivores confined to treatment areas because of territoriality or range limitations. Trace amounts of oil may cling to the feathers of birds foraging in treated areas and may be transmitted to eggs in the nest. Birds (or bird nests) accidentally sprayed during application are likely to experience mortality or reduced reproductive success. Small mammals (e.g., rodents) may experience sublethal effects because of contaminated forage, or may be killed after abandoning home ranges or territories in the treated area. Hypothermia may occur after the loss of insulating capacity of fur or feathers.

Use of diesel No. 2 in directed (selective) application techniques is not likely to adversely affect personnel, the public, nontarget vegetation, large mammals, or aquatic resources. Adverse impacts on birds and small mammal populations are likely to be insignificant.

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DRIFT CONTROL ADDITIVES

The drift of any particulate material released in the air is first and foremost a function of the particle size (Akesson et al. 1981). Inert (nontoxic) ingredients can be added to herbicide sprays to reduce drift and to improve the efficiency of herbicide use in vegetation management. When droplets between 500 and 1,000 microns in diameter are produced, the spray has virtually no drift hazard (Klingman and Ashton 1975) when applied during weather conditions specified in BPA's application guidelines.

BPA requires that aerial applications meet aerial spray delivery specifications. At a prescribed altitude in calm air conditions, the applicator must deposit a certain minimum amount of droplets within the spray zone while depositing no droplets at a distance of 10 feet beyond the edge of the spray zone. The addition of thickening agents to herbicide formulations is one means to meet these spray delivery specifications. These additives act to enlarge spray droplet or particle size and thereby control drift of herbicide spray from an immediate application site. Herbicide drift represents wasted spray as well as a potential route for herbicide damage to nontarget organisms.

Technical references on the use and efficacy of spray additives in controlling drift include: Aerial Applicator (1981), Akesson et al. (1981), Butler et al. (1969) and Yates et al. (1976). The use of Norbak and Nalco-trol (an agent identical to Lo-Drift) have been described by Butler et al. (1969) and Yates et al. (1976). Based upon the physical behavior of sprays containing these additives, the additives were judged to effectively reduce drift when used as directed. As such, the spray additives are valuable tools to help minimize the environmental impact of broadcast herbicide spraying.

NORBAK

Chemical Identification

The earliest material used as an inert spray additive for herbicide applications was Norbak, a product of the Dow Chemical Company. Norbak is a granular water-soluble plastic polyvinyl alcohol; there are no other ingredients in the commercial product. When mixed with an herbicide solution, Norbak produces a thick granular-appearing mixture

that can be pumped and sprayed without adversely affecting herbicidal action.

The water solubility of the polymer is important to its use as a particulating agent to reduce pesticide spray drift.

Polyvinyl alcohol is extensively used in commerce in the United States and abroad (Lindemann 1971). Primary uses are warp-sizing in textiles, emulsifiers, water-soluble adhesives, paper coatings and in production of other polymeric materials such as vinylacetals in safety glass. Very small amounts are used in agriculture.

Action in Vegetation

Norbak has no herbicidal activity in vegetation.

Utilization by BPA

BPA has used Norbak as a particulating agent in aerial foliage sprays of Tordon 101 (i.e., picloram and 2,4-D). Application rates of Norbank in Tordon 101 mixtures are 1-3 lbs. per acre. BPA has used an average of approximately 1,000 lbs. of Norbak each year.

Chemical Fate and Distribution in the Environment

The Norbak polymer contains carbon, hydrogen and oxygen and intermediate degradation products may include acetic acid and acetone. Polyvinyl alcohols will not persist in the environment due to their ultimate degradation to carbon dioxide and water. Studies of the environmental fate of Norbak have not been published in the literature.

Chemical Toxicology in Animals and Humans

Norbak, a particulating agent, has a low order of acute toxicity. The oral LD50 for rats was 3,250 mg/kg. Norbak was slightly irritating to the eye and not irritating to the skin at exposures that simulated conditions of normal use. Skin absorption did not result in toxicity. The dermal LD50 for rabbits was greater than 3,980 mg/kg.

In the event of accidental exposure of workers, water effectively removes Norbak from the skin, clothing or exposed surfaces (Dow Chemical Co. 1978).

Since Norbank is a spray additive rather than an active ingredient (herbicide), chronic toxicity testing has not been conducted.

Potential Impact on Nontarget Organisms

Due to the low acute toxicity of Norbank and its assumed rapid degradation in the environment, potential impacts on nontarget organisms are negligible.

Hazard Assessment for Current Use Practices

Norbak itself, diluted and used as recommended, presents no significant health hazard to applicators, the public or the environment. Herbicide mixtures containing Norbank must be removed from work areas (steps, walks or equipment) due to the physical hazard presented by the slipperiness of the formulation.

LO-DRIFT AND NALCO-TROL

Chemical Identification

Lo-Drift spray additive is also an herbicide spray additive used to increase droplet size and thereby to reduce drift. Lo-Drift is a product of Amchem Products, Inc. (Ambler, PA). The EPA Reg. No. is 264-50004AA. The principal functioning agent (30%) of Lo-Drift is acrylamide-acrylic acid copolymer made from acrylamide and acrylic acid. The remaining 70% of the product consists of agents not effective as spray adjuvants. Nalco-trol is an identical product manufactured by Nalco Chemical Co.

Acrylamide is the vinyl monomer used in the manufacture of polyacrylamides. Over 70 million pounds of acrylamide are manufactured each year for use in wastewater treatment technologies and as paper strengtheners, grouting agents, gels and adhesive agents (U.S. Department of Commerce 1976). The use of polyacrylamide-acrylic acid polymer as an herbicide spray adjuvant to reduce drift is a minor use of this versatile industrial chemical.

Action in Vegetation

Lo-Drift and Nalco-trol exhibit no herbicidal activity.

Utilization by BPA

BPA used Lo-Drift or Nalco-trol with aerial or ground-based broadcast applications of Banvel 4-W.S., Banvel 720, and Weedone 170. Up to 0.25 gallon of these spray additives is mixed with 100 gallons of herbicide mixture and applied per acre. Over the last 6 years, BPA has used an average of 580 gallons of these additives per year.

Chemical Fate and Distribution in the Environment

Polyacrylamide-acrylic acid copolymers are degraded in the environment by the action of microorganisms, by water (hydrolysis) and by other unknown factors. Degradation involves cleavage of the amide group. As a result, acrylamide monomer is not formed (Morris and Penzenstadler 1978). Polymeric fragments of the copolymer are formed as intermediates in the course of polyacrylamide degradation (Morris and Penzenstadler 1978). No other information is available on the fate of the chemical in the environment.

Chemical Toxicology in Animals and Humans

Since these additives lack herbicidal activity, they are not classed as active ingredients, and therefore they are not tested in the same manner as toxic substances.

Only acrylamide monomer is neurotoxic (Garland and Patterson 1967). No evidence is available demonstrating toxicity of the copolymer form. Signs of neurotoxicity include fatigue, mental confusion, ataxia, numbness and profuse sweating of the extremities. Workers who are engaged in the manufacture of acrylamide and in the polymerization process are at highest risk to toxic exposure. Occupational illnesses resulting from acrylamide exposures have been reviewed by Spencer and Schaumberg (1974). Only acrylamide-acrylic acid copolymer is used as a spray additive. Exposures of workers, the public and the environment to monomeric acrylamide will not occur as a result of the use of Lo-Drift spray additive.

Potential Impact on Nontarget Organisms

No impacts are expected due to low acute toxicity and its degradation into nontoxic constituents.

Hazard Assessment for Current Use Practices

Detailed instructions for the use of Lo-Drift are given on the label. An important note is the following: "Remember, herbicide drift is no accident. Common sense and sound application technology must be followed when spraying herbicides. Lo-Drift spray additive will control, but not totally eliminate, drift." The label also notes the following safety precautions: "Harmful if swallowed. Avoid contact with skin, eyes or clothing (Amchem, no date)."

Lo-Drift and Nalco-trol present no significant health hazard to applicators, the public and the environment when diluted and used as recommended. Herbicide mixtures containing Lo-Drift or Nalco-trol must be removed from walkways, floors or equipment due to the physical hazard presented by the slipperiness of the formulation.

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GLOSSARY OF TERMS FOR HERBICIDE BACKGROUND STATEMENTS

Acetonemia: a condition characterized by large amounts of acetone bodies in the blood. Acetone is a chemical compound which increases in level in blood under certain conditions such as starvation and diabetic attacks.

Acetylcholinesterase: an enzyme which breaks down actylcholine, a compound which induces nerves to transmit an electrical signal and thereby controls nerve activity.

Adenoma: a benign tumor of glandular origin and structure.

Amphipods: any of a large group of small crustaceans with a laterally compressed body (e.g. beachhoppers, scud).

Anemia: a condition marked by significant decreases in hemoglobin concentration and in the number of circulating red blood cells. Also known as oligochromemia.

Anorexia: loss of appetite.

Anthocyanin: any of the intensely colored sap soluble glycaside plant pigments responsible for most scarlet, purple, mauve and blue coloring in higher plants.

Anticholinesterase: any agent, such as a nerve gas, that inhibits the action of cholinesterase and thereby destroys or interferes with nerve conduction.

Aromatic: a group of organic compounds based on a ring of six carbon molecules bonded with a unique type of hybrid bond known as a benzene bond. The basic aromatic compound is benzene (C_6H_6). Aromatics have unique chemical properties, including a tendency to have a noticeable aroma.

Aromatic amino acid: an organic acid containing at least one amino group and one or more aromatic groups, for example, phenylalanine, one of the essential amino acids.

Aromatic hydroxylation: hydroxylation of an aromatic compound (see hydroxylation).

Aromatic nitrile: nitrile, cyanide derived by removal of water from an acid amide, associated with an aromatic compound.

Ataxia: lack of muscular coordination due to any of several nervous system diseases.

Atrophy: diminution in the size of a cell, tissue or organ that was once fully developed and of normal size.

Bioaccumulate: the process of a plant or animal selectively taking in or storing a persistent substance. Over a period of time, a higher concentration of the substance is found in the organism than in the organism's environment.

Bioassay: a method for quantitatively determining the concentration of a substance by its effect on the growth of a suitable animal, plant or microorganism under controlled conditions.

Biochemical Oxygen Demand (BOD): the amount of dissolved oxygen required to meet the metabolic needs of anaerobic microorganisms in water rich in organic matter, such as sewage.

Bioconcentration factor: a numerical value expressing the increase in concentration of a persistent substance between food chain levels, for example, the concentration of a persistent substance in the tissue of a cow may be three times that in the tissue of the grass the cow eats.

Biosynthesis: production by synthesis of chemical compound by a living organism.

Biotransformation: the process of altering a substance by reaction with metabolic processes or compounds in bacteria, plants, or animals.

Clavarium: a skull lacking facial parts and the lower jaw.

Carcinogen, carcinogenic, carcinogenicity: a substance producing or inciting cancer.

Casein: the protein of milk, a white solid soluble in acids.

Catalase: an enzyme that catalyzes the decomposition of hydrogen peroxide into molecular oxygen and water.

Cation: a positivly charged atom or group of atoms.

Chlorosis: a disease condition of green plants seen as yellowing of green parts of the plant.

Choroid plexus: any of the highly vascular, folded processes that project into the third, fourth, and lateral ventricles of the brain.

Compensatory red blood cell formation: a physiological response occurring when any one of a number of events results in a sustained lowering of oxygen levels in the bloodstream. The body may compensate by forming more red blood cells, thereby increasing the ability of the blood stream to transport oxygen.

Congestion: an abnormal accumulation of fluid, usually blood, but occasionaly bile or mucus, within the vessels of an organ or part.

Conjugate: to become joined together.

Conjunctiva: the mucous membrane covering the eyeball and lining of the eyelids.

Conjunctivitis: inflammation of the conjunctiva.

Conjunctival sac: the corner of the eye near the nose where tear ducts have their openings.

Cornea (corneal injury): the transparent anterior portion of the outer coat of the vertebrate eye covering the iris and the pupil.

Cyanosis: a bluish coloration in the skin and mucous membranes due to deficient levels of oxygen in the blood.

Cytogenetics, cytogenetic tests: a branch of molecular pathology combining the methods of cytology and genetics.

Cytoplasmic vacuoles: a membrane-bound cavity within a cell. Contents of the vacuole varies with cell type and function.

Dealkylize, dealkylation: the removal of an alkali moiety.

Deaminate, deamination: removal from a molecule of the amino group.

Decarboxylate, decarboxylation: to remove the carboxyl radical especially from amino acids and protein.

Degeneration: deterioration of a tissue or an organ in which its function is diminished or its structure is impaired.

Degenerative: relating to or tending to cause degeneration.

Degradation: conversion of an organic compound to one containing a small number of carbon atoms.

Demethylation: removal of the methyl group from a chemical compound.

Dermal: pertaining to the dermis.

Dermis: the deep layer of the skin, a dense connective tissue richly supplied with blood vessels, nerves and sensory organs.

Dermatitis: inflammation of the skin.

Detoxification: the act or process of removing a poison or the toxic properties of a substance in the body.

Dimethyl phthalate: odorless, colorless liquid, boiling at 282°C, soluble in organic solvents, slightly soluble in water, used as a plasticizer, in resins, lacquers and perfumes.

DNA repair-proficient: an organism containing the normal complement of enzymes which repair minor damage to the DNA molecule.

Dioxin (polychlorinated dibenzo-p-dioxin): PCDDs are a class of organic chemicals, including 75 isomers, each of which possess distinct physical, chemical and biological properties. One specific PCDD isomer, 2,3,7,8-tetrachlorodibenzo-d-dioxin (2,3,7,8-TCDD) has been studied most extensively. This compound and presumably other PCDDs are expected to be relatively persistent in the environment if released into air, water, and soil media.

Dyspnea: difficult or labored breathing.

Edema: an excessive accumulation of fluid in the cells, tissue spaces or body cavities due to a disturbance in the fluid exchange mechanism. Also known as dropsy.

Electromyography: a medical specialty concerned with the production and study of an electromyogram, a graphic recording of the electrical response of a muscle to stimulation.

Engorge: to devour to the limit of capacity, thus, to swell up.

Epicardium: the outer serous layer of the heart.

Epidemiology, epidemiological studies: the study of the mass aspects of disease.

Epidermis: the outer nonsensitive, nonvascular portion of the skin comprising two strata of cells, the stratum corneum and the stratum germinativum.

Epinasty: growth changes in which the upper surface of a leaf grows, thus bending the leaf downward.

Epithelium (epithelial surface): a primary animal tissue, distinguished by cells being close together with little intercellular substance, covers free surfaces and lines body cavities and ducts.

Erythrocyte: red blood cell.

Erythropoiesis: the process by which erythrocytes (red blood cells) are formed.

Fibroblasts: a stellate connective tissue cell found in fibrous tissue. Also known as a fibrocyte.

Flash point: the lowest temperature at which vapors from a volatile liquid will ignite momentarily upon the application of a small flame under specified conditions; test conditions can be either open- or closed-cup.

Focal follicular cell hyperplasia: an abnormal increase in the number of follicular cells in the thyroid gland. Follicular cells in the thyroid regulate metabolic rate and iodine levels in the blood.

Focal light cell hyperplasia: an abnormal increase in the number of "light cells" (parafollicular cells) in the thyroid. The "light cells" produce thyrocalcitonin, a hormone which lowers the concentration of calcium in the blood by inhibiting bone resorption.

Friable: easily crumbled or pulverized.

Gastritis: inflammation of the stomach.

Gastrointestinal tract: the stomach and intestine.

Hematocrit: the volume, after centrifugation, occupied by the cellular elements of blood, in relation to the total volume.

Hematology, hematological: the science of the blood, its nature, functions and diseases.

Hematuria: a pathological condition in which the urine contains blood.

Hemoglobin: The iron-containing, oxygen-carrying molecule of the red blood cells of vertebrates.

Hemosiderosis: deposition of hemosiderin, an iron-containing compound, in body tissues without tissue damage, reflecting an increase in body iron stores.

Histidine: a crystalline basic amino acid present in large amounts in hemoglobin and resulting from the hydrolysis of most proteins. This amino acid is present in most organisms and influences the mechanism controlling gene expression.

Histidine reverse mutation: some bacteria require histidine in the environment in order to survive. Other bacteria do not. Certain compounds may cause mutations which reverse the need for or no need for histidine.

Histopathology: a branch of pathology that deals with tissue changes associated with disease.

Humic: relating to decomposition-resistant organic matter in soil.

Hydrolysis: decomposition or alteration of a chemical substance by water.

Hydroxylation, hydroxylated: one of several types of reactions used to introduce one or more hydroxyl groups into organic compounds; an oxidation reaction. Opposed to hydrolysis.

Hyperemia: an excess of blood within an organ or tissue caused by blood vessel dilation or impaired drainage especially of the skin.

Hyperplasia: increase in cell number causing an increase in the size of a tissue or organ.

Hypertrophy: increase in cell size causing an increase in the size of an organ or tissue.

Hypothermia: condition of reduced body temperature in warm-blooded animals.

Intestinal mucosa: the mucous membrane of the tubular portion of the vertebrate digestive tract, usually between the stomach and the cloaca or anus.

Intraperitoneal, intraperitoneally: related to a structure or process occurring within the peritoneum, a membranous lining of the body cavity.

Isopods: any of a large order of small sessile-eyed crustaceans with the body composed of seven free thoracic segments, each bearing a pair of similar legs (e.g. sowbugs, pillbugs).

K-value: the ratio of the amount of herbicide absorbed on soil (ppm) to an equilibrium concentration of 1 ppm in water.

LC50 (lethal concentration 50): abbreviation denoting the lethal concentration or concentrations of toxicant necessary to kill 50% of the organisms being tested.

LD50 (lethal dose 50): the dose of a substance which is fatal to 50% of the test animals. Also known as median lethal dose.

Lesion: a structural or functional alteration due to injury or disease.

Lignin: a substance that together with cellulose forms the woody cell wall of plants and cements them together.

Lymphatic system: a system of vessels and nodes conveying lymph in the vertebrate body, beginning with capillaries in tissue spaces and eventually forming the thoracic ducts which empty into the subclavian veins.

Lysimeter: an instrument for measuring the water percolating through soils and determining the materials dissolved by the water.

Median threshold limits (TLm): synonymous with the median tolerance limit (TL50) but expressed in a slightly different way, i.e. the concentration of a test material at which half of the test animals are able to survive under test conditions over a specified time.

Mesophyll: parenchymatous tissue between the upper and lower epidermal layers in foliage leaves.

Metabolism: the physical and chemical processes by which foodstuffs are synthesized into complex elements, complex substances are transformed into simple ones, and energy is made available for use by an organism.

Metabolite: A product of intermediary metabolism.

Methemoglobinemia: the presence of hemoglobin (red blood cell pigment) in the oxidized state in the blood. Hemoglobin is normally oxygenated in the blood.

Midrib: the large central vein of a leaf.

Miscible: tendency of two or more liquids to form a uniform blend.

Mitosis: nuclear division involving exact duplication and separation of the chromosome threads so that each of the two daughter nuclei carries a chromosome complement identical to that of the parent nucleus.

Moiety (hydroxy moiety): a part or portion of a molecule generally complex, having a characteristic chemical or pharmacological property.

Myotononia: tonic muscular spasm occurring after injury or infection.

Necropsy: to perform an autopsy.

Necrosis, necrotic: death of a cell or group of cells as a result of injury, disease or other pathologic state.

Neuropathy: any disease affecting neurons.

Nitrate reductase: an enzyme which transforms nitrate into nitrite.

Oncology: the study of the causes, development, characteristics and treatment of tumors.

Organophosphate (organophosphate insecticides): a chemical compound characterized by the presence of organic phosphate esters, often useful for providing phosphorous to deep root systems.

Organoleptic, organoleptically: related to use of one or more organs of special sense, e.g. taste, smell.

Ossification: process of forming bone.

Parenchyma: the specialized epithelial portion of an organ, as contrasted with the supporting connective tissue and nutritive framework.

Paresthesia: tingling, crawling or burning sensation of the skin.

Parturition: process of giving birth (labor).

Petechiae: hemorrhages the size of the head of a pin.

Photodecomposition: the process of light energy causing cleavage of chemical bonds within a compound, resulting in formation of two or more smaller, different compounds.

Photooxidation: process occurring when light energy causes alteration of a chemical bond within a compound, resulting in either addition of an oxygen atom or loss of a hydrogen atom.

Photosynthate: any chemical product, usually a carbohydrate, resulting from photosynthetic activity in a plant.

Phytotoxin, phytotoxic, phytotoxicity: a substance toxic to plants.

Point mutation: mutation of a single gene due to addition, loss, replacement or change of sequence in one or more pairs of the deoxyribonucleic acid of that gene.

Postemergence: applied after emergence of the specified weed or planted crop.

Postimplantation: the period of time after embryos become implanted in the wall of the uterus.

Preemergence: applied prior to emergence of the specified weed or planted crop.

Proliferative growth: to grow by rapid production of new parts, cells, buds or offspring.

Propazine: a colorless solid with a melting point of 212-214^{o}C; used as a preemergence herbicide for control of weeds in milo and sweet sorghum.

Pulmonary edema: an effusion of fluid into the alveoli and interstitial spaces of the lungs.

Recombination: the occurrence of gene combinations in the progeny that differ from those of the parents as a result of independent assortment linkage and crossing over.

Ring cleavage: a reaction which causes a break in chemical bonds in a compound which originally was organized with some of the elements bonded into a ring formation.

Rumen: first chamber of the ruminant (e.g., cow) stomach.

Side chain: a grouping of similar atoms (two or more, generally carbons, as in ethyl radical, C_2H_5-) that branches off from a straight chain or cyclic (for example, benzene) molecule.

Soil colloids: colloidal complex of soils composed principally of clay and humus.

Soil:herbicide ratio: the relationship between the weight of soil to the weight of herbicide.

Soil:water partition coefficients: the ratio of the amount of herbicide absorbed on soil and dissolved in groundwater at equilibrium (see K-value).

Spray interception disc: a small disc placed in the path of a spraying operation and used to capture spray particles in order to measure the amount of spray reaching the target area.

Subacute: moderately acute.

Subcutaneous tissue: the layer of loose tissue beneath the dermis.

Sublimation: the process by which solids are transformed directly to the vapor state or vice versa without passing through the liquid phase.

Sulphaemoglobin: a greenish substance derived from hemoglobin by the action of hydrogen sulfide, it may appear in the blood following the ingestion of sulfanilamide and other substances.

Surfactant: a soluble compound that reduces the surface tension of liquids or reduces interfacial tension between two liquids or a liquid and a solid. Also known as a surface-active agent.

Teratogen: an agent causing formation of a congenital anamoly or monstrosity.

Teratogenesis: the formation of a fetal monstrosity.

Teratology: the science of fetal malformations and monstrosities.

Triazine: any of three compounds $C_3H_3N_3$, containing a ring composed of three carbon and three nitrogen atoms.

Tryptophan mutation system: an amino acid obtained from casein, fibrin and certain other proteins. It is a precursor of indoleacetic acid, serotonin and nicotinic acid. It is an essential amino acid. Certain mutant bacteria are incapable of making it, and require tryptophan in the culture medium.

Tymponites: a distension of the abdomen caused by accumulation of gas in the intestinal tract or peritoneal cavity.

Tympany: swelling from abdominal gas.

Tyvek fiber: a type of inert material used as a carrier in testing chemicals applied to the skin.

Uracil: a pyrimidine base important as a component of ribonucleic acid.

Urea: A natural product of protein metabolism found in urine, synthesized as white crystals or powder with a melting point of 132-7^{o}C, soluble in water, alcohol and benzene, used as a fertilizer in plastics, adhesives, flameproofing agents and in medicine.

Vacuolization: process of forming vacuoles (membrane-bound cavities) within a cell.

Vermiculite: a clay mineral constituent similar to chlorite or montmorillonite, and consisting of trioctahedral mica sheets separated by double water layers.

HERBICIDES APPROVED FOR USE BY BPA

APPENDIX B

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APPENDIX B

Additional information on herbicides approved by BPA and proposed for use in its vegetation management program:

Table B-1: Commercial Herbicide Products

Table B-2: Application Specifications

Table B-1. Commercial Herbicide Products Approved by BPA for Use in its Vegetation Management Program[1]

Common Name	Trade Name[2]	Manufacturer	EPA Registration Number	Formulation[3]	Ingredients	
Ammonium sulfamate		DuPont	352-206-AA	WSC	Active ingredient:	Ammonium sulfamate.....95.0%
	Ammate® X-NI	DuPont	352-311	WSC	Inert ingredients:	.....5.0%
Atrazine	Aatrex® 80W	Ciba-Geigy	100-439	WP	Active ingredient:	Atrazine.....76.0%
					Related compounds:	.....4.0%
					Inert ingredients:	.....20.0%
	Aatrex® Nine-0®	Ciba-Geigy	100-585	WDG	Active ingredient:	Atrazine.....85.5%
					Related compounds:	.....4.5%
					Inert ingredients:	.....10.0%
Bromacil	Hyvar® X-L	DuPont	352-346-ZA	WSL	Active ingredient:	Bromacil.....21.9%
					Inert ingredients:	.....78.1%
Bromacil + Diuron	Krovar® I	DuPont	352-352-AA	WP	Active ingredients:	Bromacil.....40.0%
						Diuron.....40.0%
					Inert ingredients:	.....20.0%
Dicamba	Banvel® 4-0.S.[4]	Velsicol	876-156-AA	OSL	Active ingredients:	Dicamba.....44.72%
					Related acids:	.....6.68%
					Inert ingredients:	.....48.60%
	Banvel® 4-W.S.	Velsicol	876-159-AA	WSL	Active ingredients:	Dicamba.....49.0%
					Related acids	.....7.9%
					Inert ingredients:	.....43.1%
	Banvel® XP[4]	Velsicol	876-178-AA	P	Active ingredient:	Dicamba.....10.0%
					Related ingredients:	.....1.8%
					Inert ingredients:	.....88.2%
	Banvel® 5G	Velsicol	876-103-AA	G	Active ingredient:	Dicamba.....5.0%
					Related acids	.....0.9%
					Inert ingredients:	.....94.1%

Table B-1. (Cont'd.)

Common Name	Trade Name[2]	Manufacturer	EPA Registration Number	Formulation[3]	Ingredients
Dicamba + 2,4-D	Banvel®-720	Velsicol	876-177-AA	WSL	Active ingredients: Dicamba.....12.90% Dimethylamine salts of related acids.....1.80% Dimethylamine salt of 2,4-Dichlorophenoxyacetic acid*..25.80% Inert ingredients: .....59.50% *Equivalent to 21.43% 2,4-Dichlorophenoxyacetic acid
Dichlobenil	Casoron® G-4	Thompson-Hayward	148-614	G	Active ingredient: Dichlobenil.....4.0% Inert ingredients: 96.0%
Glyphosate	Roundup®	Monsanto	524-308-AA	WSL	Active ingredient: Isopropylamine salt of glyphosate .....41.0% Inert ingredients: 59.0%
Monuron	Telvar®[4] Monuron 80 WP	DuPont Aceto Agric. Chem.	352-246 (USDA No.) 2749-60	WP	Active ingredient: Monuron.....80.0% Inert ingredients: 20.0%
Picloram	Tordon® 10K Amdon® 10K	Dow Union Carbide (Amchem)	464-320 464-320AA-264	P P	Active ingredient: Picloram as the potassium salt*...11.6% Inert ingredients: .....88.4% *Acid equivalent of 10% Picloram
Picloram + 2,4-D	Tordon® 101 Amdon® 101	Dow Union Carbide (Amchem)	464-306 464-306AA-264	WSL WSL	Active ingredients: Picloram as the triisopropanolamine salt*.....10.2%. 2,4-Dichlorophenoxyacetic acid as the triisopropanolamine salt* .....39.6% Inert ingredients: .....50.2% *Acid equivalents of 5.7% Picloram and 21.2% 2,4-Dichlorophenoxyacetic acid

Table B-1. (Cont'd.)

Common Name	Trade Name[2]	Manufacturer	EPA Registration Number	Formulation[3]	Ingredients
Prometon	Pramitol® 25E	Ciba-Geigy	100-443AA	EC	Active ingredient: Prometon.....25.0% Inert ingredients: 75.0%
Prometon + Simazine + Sodium chlorate + Sodium metaborate	Pramitol® 5PS	Ciba-Geigy	100-479	P	Active ingredients: Prometon.....5.00% Simazine .....0.75% Sodium chlorate .....40.00% Sodium metaborate .....50.00% Inert ingredients: 4.25%
Sodium + chlorate Sodium metaborate + Bromacil	Oxy Ureabor®	Occidental	10659-51	G/WSC	Active ingredients: Sodium metaborate tetrahydrate*...66.5% Sodium chlorate .....30.0% Bromacil .....1.5% Inert ingredients: .....2.0% *Boron trioxide equivalent of 22.6%
Tebuthiuron	Spike® 1G	Elanco	1471-104	G	Active ingredient: Tebuthiuron.....1.0% Inert ingredients: 99.0%
	Spike® 5G	Elanco	1471-103	G	Active ingredient: Tebuthiuron.....5.0% Inert ingredients: 95.0%
	Spike® 80W	Elanco	1471-97	WP	Active ingredient: Tebuthiuron.....80.0% Inert ingredients: 20.0%
2,4-D amine	Formula 40®	Dow	464-1-AA	WSA	Active ingredient: Alkanolamine salts of 2,4-Dichlorophenoxyacetic acid*.....59.7% Inert ingredients: .....40.3% *Acid equivalent of 38.6% 2,4-Dichlorophenoxyacetic acid
2,4-D ester	Esteron® 99® Concentrate	Dow	464-201	LVE	Active ingredient: 2,4-Dichlorophenoxyacetic acid as propylene glycol butyl ether esters*.....72.8% Inert ingredients: .....27.2% *Acid equivalent of 44.9% 2,4-Dichlorophenoxyacetic acid

Table B-1. (Cont'd.)

Common Name	Trade Name[2]	Manufacturer	EPA Registration Number	Formulation[3]	Ingredients
2,4-DP + 2,4-D	Weedone® 170	Union Carbide (Amchem)	264-222-ZB	LVE	Active ingredients: 2,4-Dichlorophenoxypropionic acid as butoxyethanol ester*.....31.6% 2,4-Dichlorophenoxyacetic acid as butoxyethanol ester..32.2% Inert ingredients: .....36.2% *Acid equivalent of 22.2% 2,4-Dichlorophenoxypropionic acid and 22.2% 2,4-Dichlorophenoxyacetic acid

[1]This is a list of commercial herbicide products registered by EPA as of May 1, 1983. Since registrations change periodically, be sure to consult the most current label for each product to ensure it is still registered for the intended use. This list does not preclude the use of other herbicide products with the same ingredients if registered for use in situations appropriate to the BPA vegetation management program. Furthermore, all of the herbicides listed in this table may not necessarily be used in a given fiscal year program.

[2]All trade names are trademarks registered by the respective manufacturers. Products listed in this table are intended to serve as examples of materials currently used by BPA as of May 1, 1983. This list does not endorse the use of such products to the exclusion of other brands or formulations if registered and appropriate to the BPA vegetation management program.

[3]Abbreviations stand for the following types of formulations:

EC = Emulsifiable concentrate
G = Granule
LVE = Low volatile ester
OSL = Oil soluble liquid
P = Pellet
WDG = Water dispersable granule
WP = Wettable powder
WSA = Water soluble amine
WSC = Water soluble crystals
WSL = Water soluble liquid

[4]No longer manufactured as a commercial product.

Table B-2. Application Specifications for Use of Herbicide Products by BPA in its Vegetation Management Program[1]

Common Name	Trade Name[2]	Application Mixture	Herbicide Placement	Labeled Rates of Application Used by BPA	Time of Application
Ammonium sulfamate	Amate® X-NI	Aqueous mixture; formulated product	Foliage and stems	Foliage spray: aqueous spray, 60 lb product/100 gal spray	Full leaf stage until leaf discoloration begins
			Stem frills, notches or injections	Cut-stump treatment: sprinkle crystals liberally on freshly cut surface, or spray stump thoroughly with a solution made from 7-10 lb product in 2 gal water	As soon after cutting as possible
			Cut stump surface	Single-stem treatments: saturate frilled area with a solution made from 7-10 lb product in 2 gal water; alternatively, apply 0.5 oz of product crystals per notch spaced 2-6 in. apart	As above
Atrazine	Aatrex® 80W	Aqueous mixture	Surface of soil and vegetation	Apply following amounts in a minimum of 1 gal spray/lb product:	Just before or soon after weeds begin growth
				Annual herbaceous weeds: 6-12 lb product/A	
				Hard-to-kill annual herbaceous weeds and many perennial herbaceous weeds: 12.5-25 lb product/A	
				Hard-to-kill biennial and perennial weeds: 25-50 lb product/A	
	Aatrex® Nine-O®	Aqueous mixture	Surface of soil and vegetation	Apply following amounts in a minimum of 1 gal spray/lb product:	Just before or soon after weeds begin growth
				Annual herbaceous weeds: 5.3-11.1 lb product/A	
				Hard-to-kill annual herbaceous weeds and many perennial herbaceous weeds: 11.1-22.2 lb product/A	
				Hard-to-kill biennial weeds: 22.2-44.4 lb product/A	
Bromacil	Hyvar® X-L	Formulated product	Soil surface	Brush control: undiluted, 5-10 ml/stem 2-4 in. in basal diameter; directed with an exact delivery hand-gun applicator to the root collar	Just before or during period of active plant growth (spring or summer)
Bromacil + Diuron	Krovar® I	Aqueous mixture	Soil surface	Short-term control of annual weeds: 4-6 lb product/A in 40-100 gal water	Just prior to weed emergence or in early stages of weed growth

Table B-2. Cont'd.

Common Name	Trade Name[2]	Application Mixture	Herbicide Placement	Labeled Rates of Application Used by BPA	Time of Application
				Extended control of annual weeds and partial control of perennial herbaceous weeds: 7-40 lb product/A in 40-100 gal water	
Dicamba	Banvel® 4-O.S.	Oil mixture	Stems	Mix 1-3 qt product with 2-6 lb a.e. 2,4-D oil soluble ester in sufficient amount of diesel oil or fuel oil to make 100 gal of spray mixture, spray the lower 1.5-2 ft of bole to point of runoff	Basal-stem treatment done year-round
	Banvel® 4-W.S.	Aqueous mixture	Foliage and stems Stem frills, notches or injections Cambium of cut stump	Foliage-stem spray for woody brush: 1 qt product + 0.5 gal (2 lb a.i.) 2,4-D WSA or emulsifiable LVE in 99.25 gal water; apply to point of runoff from foliage, stem and root crown (150-300 gal/A)	After leaves are fully developed and until 3 weeks before frost
				Single-stem treatment for hardwoods: mix 1 part product with 1 part water and apply 0.5-1 ml of solution to each notch or injection spaced up to 2 in. apart	Anytime of year
				Aerial application: 5 gal product + 10 gal (40 lb a.i.) 2,4-D WSA or emulsifiable LVE in 85 gal water; apply at 15 gal spray mix/A; alternatively, mix 2.5 gal product + 5 gal (20 lb a.i.) 2,4-D WSA or emulsifiable LVE in 92.5 gal water and apply at 30 gal spray mix/A	After leaves are fully developed and until 3 weeks before frost
	Banvel® XP[3] Banvel® 5G	Formulated product	Soil surface beneath canopy	Apply as a directed application at rates equivalent to 80-160 lb/A	Just prior to or in the early part of the rainy season
Dicamba + 2,4-D	Banvel® 720	Aqueous mixture	Foliage and stems Foliage	Foliage-stem spray for woody brush: 1 gal product in 99 gal water; apply to point of runoff on stems, foliage and root crowns (100-300 gal/A)	After leaves are fully developed and until 3 weeks before frost

Table B-2. Cont'd.

Common Name	Trade Name[2]	Application Mixture	Herbicide Placement	Labeled Rates of Application Used by BPA	Time of Application
				Aerial application for woody brush: 3 gal product in 12-27 gal water applied at 15-30 gal/A, respectively	As above
				Foliage spray for annual and perennial broadleaf weeds (e.g., noxious weeds): 0.5-1 gal product in 99.5 or 99 gal water, respectively; apply at rate of 100 gal/A	When weeds are actively growing
Dichlobenil	Casoron® G-4	Formulated product	Soil surface	Annual weeds: 100-150 lb product/A	Early spring, prior to germination
				Perennial weeds; 150-200 lb product/A	Late fall from November 15 to February 15
Glyphosate	Roundup®	Aqueous mixture	Foliage	Foliage spray: 1-2% solution (4-8 qt/100 gal water); apply until plant foliage is wet but not to point of runoff	Late in the growing season or after plants have reached maturity
Monuron	Telvar®[3] Monuron 80 WP	Aqueous mixture	Soil surface	Annual weeds: 5-20 lb product/A	Best results when applied shortly before weed growth begins
				Annual and perennial weeds: 20-60 lb product/A	
				Apply above amounts in enough water to uniformly cover the area	
Picloram	Tordon® 10K Amdon® 10K	Formulated product	Soil surface	Broadcast: 20-85 lb product/A (depends on species and density	Anytime soil not frozen; best results in spring before growth begins or during periods of vigorous growth when subsequent rainfall can be expected
				Directed: 3 oz/100 sq ft soil surface; apply uniformly beneath crown	
Picloram + 2,4-D	Tordon® 101 Amdon® 101	Aqueous mixture; formulated product	Foliage and stems	Broadleaf herbaceous weeds: 0.5-3 gal product/A	
				Woody plants and vines: 1-3 gal product/A	
				Ground foliage-stem spray: 1 gal in 100 gal aqueous spray; wet all leaves, stems and root collars	When weeds and brush are actively growing, but after foliage is well developed

Table B-2. Cont'd.

Common Name	Trade Name[2]	Application Mixture	Herbicide Placement	Labeled Rates of Application Used by BPA	Time of Application
				Aerial foliage spray: apply required amount of mixture in 10-25 gal spray/A; use in conjunction with Norbak® particulating agent or equivalent material which provides a thickened (high viscosity) spray mixture; (note: in Oregon other drift control systems [e.g., Microfoil® boom], additives, or specifications may be used as recommended by forestry herbicide specialists or representatives of the Dow Chemical Company)	As above
				Single-stem treatments: Injector use: undiluted or diluted product 1:1 with water and apply 0.5-1.0 ml, respectively, through the bark at intervals of 3 in. between edges of injector wounds, which completely surround the tree at a convenient height Girdle method: wet the cut surface with diluted solution	Any season except during periods of heavy sap flow of certain species
				Cut-stump treatment: apply undiluted or diluted product 1:1 to all the cambium of freshly-cut stumps	
Prometon	Pramitol® 25E	Aqueous mixture	Foliage and soil surface	Annuals and perennials: 15 gal product in 100 gal water/A	Prior to weed emergence or when weeds are young and actively growing
Prometon + Simazine + Sodium chlorate + Sodium metaborate	Pramitol® 5PS	Formulated product	Soils surface	Annuals: 0.5-1 lb product/ 100 sq ft Perennials: 1-2 lb product/ 100 sq ft	Before or after plant growth begins, provided there will be sufficient rainfall to move the chemical into the root zone

Table B-2. Cont'd.

Common Name	Trade Name[2]	Application Mixture	Herbicide Placement	Labeled Rates of Application Used by BPA	Time of Application
Sodium chlorate + Sodium metaborate + Bromacil	Oxy Ureabor®	Formulated product; aqueous mixture	Dry application: soil surface Spray application: foliage	Dry application: 0.5-3.0 lb product/100 sq ft Spray application: 0.5-3.0 lb product/100 sq ft; apply to thoroughly wet the foliage	During the growing season
Tebuthiuron	Spike® 1G	Formulated product	Soil surface	400-1,600 lb product/A, depending on target weeds	Best results obtained if applied shortly before or at the time plant growth begins
	Spike® 5G	Formulated product	Soil surface	80-320 lb product/A, depending on target weeds	As above
	Spike® 80W	Aqueous mixture	Soil surface	Initial treatment: 5-20 lb product/A (depending on target weeds) in 15-20 gal water/A Maintenance treatment: 1.5-3 lb product/A (depending on target weeds) in 15-20 gal water/A	As above
2,4-D amine	Formula 40®	Aqueous mixture; formulated product	Foliage and stems Stems frills, notches or injections	Broadleaf weed control: Broadcast spray: 1-2 qt product/A in the amount of water needed for uniform application; (note: can be combined with Banvel® 4-W.S., Tordon® 101, or Amdon® 101) Directed spray: 0.25 pt product in 3 gal water and spray to thoroughly wet all foliage	During warm weather when weeds are young and actively growing
				Single-stem treatment: 1-2 ml undiluted product injected at intervals of at least 1-3 in. between edges of the injector wounds and circling the trunk near the base of the tree	During any season except that maples should not be treated during the spring sap flow
2,4-D ester	Esteron® 99® Concentrate	Aqueous mixture	Foliage and stems	Broadcast treatment: Broadleaf herbaceous weeds: apply 1-3 qt product/A in enough spray to provide uniform coverage of weeds and brush (usually 5-20 gal or more spray/A for ground equipment and 3-5 gal or more spray/A for aircraft)	When weeds or brush are actively growing, up to 3 weeks before frost as long as soil moisture is sufficient for active growth of the brush

Table B-2. Cont'd.

Common Name	Trade Name[2]	Application Mixture	Herbicide Placement	Labeled Rates of Application Used by BPA	Time of Application
				Woody brush: use 3-4 qt product in 100 gal water/A and wet all parts (foliage, stems and bark); wetting agent may be added to the spray if needed for increased effectiveness (note: can be combined with Banvel® 4-W.S.)	
				Directed treatment: 0.25 pt product in 3 gal water and spray to thoroughly wet all weed foliage	
2,4-DP + 2,4-D	Weedone® 170	Aqueous or oil mixture of oil-water emulsion	Foliage and stems Basal part of stems (including root collar)	Foliage-stem treatment and broadcast spray: 1-1.5 gal product in 100 gal water; thoroughly wet all leaves, stems and suckers to ground line (200-600 gal spray/A)	From time foliage is fully developed until plants begin to go dormant
				Basal-stem treatment: 3-4 gal product in 100 gal oil; thoroughly wet the base and root collar	Can be applied in any season
				Modified basal-stem treatment	When brush is in full foliage
				Early season: 1-1.5 gal product in 10 gal diesel oil and 89 gal water	
				During dry weather or latter part of spray season: 1-1.5 gal product in 15 gal diesel oil and 83.5 gal water	
				Drench base of plants and then wet the lower 4/5 of the remaining stems and leaves to runoff	
				Cut surface treatments: mix 3-4 gal product in 100 gal oil and apply as follows to:	Soon after trees cut or frilled; any time of year
				Cut-stump: entire stump thoroughly drenched	
				Frill: pour mixture into ax frills forming a continuous ring around the trunk near the base	

Table B-2. Cont'd.

FOOTNOTES:

[1]Information presented in this is table is based on label directions and BPA use patterns as of May 1, 1983. Since product labels change periodically, the most current label should be consulted for appropriate use patterns. Furthermore, the use patterns described in this table should not preclude other use patterns if not prohibited by the label, and if they are appropriate for BPA vegetation management situations.

[2]Products listed in this table are intended to serve as examples of materials currently used by BPA as of May 1, 1983. This list does not endorse the use of such products to the exclusion of other brands or formulations if registered and appropriate to the BPA vegetation management program.

[3]No longer manufactured as a commercial herbicide product.

HISTORICAL USE

APPENDIX C

HISTORICAL USE

APPENDIX C

APPENDIX C

Tables showing average number of right-of-way acres programmed for treatment per year for 10-year period 1973-1982:

(for discussion, see Chapter 5)

Table C-1: Counties of Puget Sound Area

Table C-2: Counties of Lower Columbia Area

Table C-3: Counties of Upper Columbia Area

Table C-4: Counties of Snake River Area

Table C-5: Percentages by Method for BPA Service Area

Note: These tables indicate the geographic extent of past BPA vegetation management in counties of the BPA service area. Future vegetation management activities will be similar in overall extent but may differ with respect to types of control methods used.

Table C-1. Average Number of Right-of-Way Acres Programmed for Treatment Per Year for 10-Year Period 1973-1982 in Puget Sound Area[1]

State/County	Average Acres Per Year by Type of Control Method: Manual	Aerial (Broadcast) Herbicide	Spot Herbicide	Method Not Specified[2]	Average Acres Per Year by All Methods Combined
Washington					
Clallam	19	-	182	14	215
Grays Harbor	22	169	243	13	447
Jefferson	10	-	124	16	150
King	104	110	1,077	170	1,461
Kitsap	12	-	97	13	122
Lewis	42	411	590	20	1,063
Mason	23	-	302	-	325
Pacific	20	22	226	4	272
Pierce	23	550	285	24	882
Skagit	12	-	118	38	168
Snohomish	33	-	395	24	452
Thurston	41	365	397	9	812
Whatcom	25	-	239	23	287
TOTAL	386	1,627	4,275	368	6,656
Percent of Area Total	6	24	64	6	100

FOOTNOTES:

[1]Right-of-way vegetation management includes brush control, noxious weed control, and wood pole protection.

[2]The 1973 tabulation did not separate treatments by control method; acreages for that year are shown as "method not specified".

Table C-2. Average Right-of-Way Acres Programmed for Treatment Per Year for 10-Year Period 1973-1982 in Lower Columbia Area[1]

State/County	Average Acres Per Year by Type of Control Method: Manual	Aerial (Broadcast) Herbicide	Spot Herbicide	Method Not Specified[2]	Average Acres Per Year by all Methods Combined
Oregon					
Benton	2	23	54	4	83
Clackamas	4	116	322	52	494
Clatsop	2	-	103	93	198
Columbia	5	30	263	106	404
Coos	47	142	178	121	488
Curry	5	89	205	16	315
Douglas	22	107	185	10	324
Lane	32	201	390	69	692
Lincoln	4	64	62	23	153
Linn	15	70	329	51	465
Marion	5	69	127	32	233
Multnomah	24	29	92	12	157
Polk	5	-	47	23	75
Tillamook	20	-	164	30	214
Washington	5	13	97	10	125
Yamhill	1	-	36	3	40
Washington					
Clark	10	8	205	28	251
Cowlitz	25	216	280	36	557
Skamania	95	32	591	29	747
Wahkiakum	12	-	81	-	93
TOTAL	340	1,209	3,811	748	6,108
Percent of Area Total	6	20	62	12	100

FOOTNOTES:

[1]Right-of-way vegetation management includes brush control, noxious weed control, and wood pole protection.

[2]The 1973 tabulation did not separate treatments by control method; acreages for that year are shown as "method not specified".

Table C-3. Average Right-of-Way Acres Programmed for Treatment Per Year for 10-Year Period 1973-1982 in Upper Columbia Area[1]

State/County	Average Acres Per Year by Type of Control Method: Manual	Aerial (Broadcast) Herbicide	Spot Herbicide	Method Not Specified[2]	Average Acres Per Year by all Methods Combined
Washington					
Chelan	41	-	151	100	292
Columbia	-	-	20	-	20
Douglas	-	-	47	-	47
Ferry	10	-	5	10	25
Grant	1	-	88	-	89
Kittitas	134	65[3]	269	30	498
Lincoln	3	-	76	-	79
Okanogan	1	-	15	-	16
Pend Oreille	23	150[4]	64	5	242
Spokane	18	-	127	-	145
Stevens	1	-	37	13	51
Whitman	-	-	57	-	57
Idaho					
Bonner	10	-	83	-	93
Boundary	9	-	20	5	34
Clearwater	7	-	15	-	22
Kootenai	2	-	35	-	37
Latah	2	-	20	-	22
Nez Perce	1	-	35	-	36
Shoshone	3	-	23	-	26
Montana					
Deer Lodge	<1	-	40	-	40
Flathead	100	-	28	37	165
Granite	<1	-	8	-	8
Lake	25	-	8	-	33
Lincoln	35	-	28	5	68
Mineral	3	-	-	-	3
Missoula	1	-	15	-	16
Powell	<1	-	22	-	22
Sanders	23	-	19	15	57
TOTAL	453	215	1,355	220	2,243
Percent of Area Total	20	10	60	10	100

FOOTNOTES:

[1]Right-of-way vegetation management includes brush control, noxious weed control, and wood pole protection.
[2]The 1973 tabulation did not separate treatments by control method; acreages for that year are shown as "Method not Specified".
[3]All acres treated in single year 1976.
[4]All acres treated in single year 1974.

Table C-4. Average Right-of-Way Acres Programmed for Treatment Per Year for 10-Year Period 1973-1982 in Snake River Area[1]

State/County	Average Acres Per Year by Type of Control Method: Manual	Aerial (Broadcast) Herbicide	Spot Herbicide	Method Not Specified[2]	Average Acres Per Year by All Methods Combined[3]
Oregon					
Crook	-	-	5	-	5
Deschutes	1	-	38	1	40
Gilliam	-	-	5	-	5
Hood River	115	-	283	17	415
Klamath	5	-	52	4	61
Lake	-	-	7	-	7
Sherman	-	-	9	-	9
Umatilla	-	-	10	4	14
Union	4	-	9	4	17
Wasco	25	-	219	51	295
Washington					
Franklin	-	-	10	-	10
Klickitat	44	-	222	13	279
Walla Walla	-	-	9	-	9
Yakima	-	-	25	1	26
Idaho					
Bonneville	5	<1[4]	6	-	11
Cassia	-	<1[4]	1	-	1
Elmore	1	-	2	-	3
Fremont	1	-	3	-	4
Gem	<1	-	1	-	1
Minidoka	-	-	4	-	4
Teton	4	-	4	-	8
Wyoming					
Lincoln	<1	-	1	-	1
Teton	<1	-	<1	-	<1
TOTAL	205	-	925	95	1,225
Percent of Area Total	17	0	75	8	100

FOOTNOTES:

[1]Right-of-way vegetation management includes brush control, noxious weed control, and wood pole protection.
[2]The 1973 tabulation did not separate treatment by control method; acreages for that year are shown as "Method not Specified".
[3]These additional counties have the following average acres treated per year:
Skamania, WA 100-300 ac/yr
Multnomah, OR 10-50 ac/yr
Gallatin, MT 10-50 ac/yr
Modoc, CA 10-50 ac/yr
[4]All acres treated in single year 1974.

Table C-5. Percentages of Treated Acres by Treatment Method on BPA Rights-of-Way for Each Area for 10-Year Period, 1973-1982

Area	Treatment Method				
	Manual	Aerial (Broadcast) Herbicide	Spot Herbicide	Not Specified	Total
Puget Sound	6%	24	64	6	100
Lower Columbia	6	20	62	12	100
Upper Columbia	20	10	60	10	100
Snake River	17	0	75	8	100
Total for Entire BPA Service Area	9	19	63	9	100

LANDSCAPE TYPES

APPENDIX D

LANDSCAPE TYPES

APPENDIX D

APPENDIX D

LANDSCAPE SETTING TYPES OF THE BPA SERVICE AREA

The following paragraphs describe the general distribution, character, and visibility potential of landscape setting types within th BPA service area. These landscapes were defined originally in Jones & Jones (1976: Final Report on Measuring the Visibility of the H. V. Transmission Facilities in the Pacific Northwest. Prepared for BPA). Refer to Figure 6-2 for graphic portrayals of the 12 landscape setting types.

1. Flatland/Grassland-Shrubland -- Much of eastern Washington and Oregon and southern Idaho are included in this category. Rights-of-way are virtually indistinguishable from their surroundings, since the surrounding vegetation cover carries through the right-of-way without interruption. Visibility is unobstructed, but surface haze often tends to reduce the visibility of the right-of-way. The viewer position is generally level and hence views of the right-of-way surface are quite limited.

2. Flatland/Open Forest -- Certain areas of eastern Washington and Oregon, Idaho and Montana, where rainfall is sufficient to permit stands of juniper and ponderosa pines, are included in this category. The right-of-way vegetation contrast is only moderately visible due to the limited management required. The edge effect caused by the right-of-way is blurred because of the clump or cluster of natural vegetation as well as the continuous nature of the understory. The viewer position is level and, depending upon the density of the clusters of vegetation, the degree of visibility is reduced.

3. Flatland/Closed Forest -- Much of the Puget Sound area, coastal valleys and flatlands and the Willamette Valley are included in this category. The right-of-way vegetation contrast is highly visible due to the potential height and density of the vegetation within the closed forest category. The edge effect caused by the right-of-way is distinct due to the contrast between the dense canopy vegetation and the open right-of-way. The viewer position is level and, with the exception of views within the right-of-way, the views are screened by the dense vegetation.

4. Valley Floor/Grassland-Shrubland -- This zone is typified by the broad valleys that have been cleared west of the Cascades and the dissected areas in the intermountain region. The right-of-way is indistinguishable from the surrounding landscape, since the surrounding vegetation cover carries through the right-of-way without interruption. The visibility is unobstructed, but surface haze and reduced length of view often tend to reduce the visibility of the right-of-way. The viewer position is level and hence the view of the right-of-way surface is quite short. Views of the right-of-way can be obtained from the adjacent hillsides and ridgelines.

5. Valley Floor/Open Forest -- This zone is typified by the valleys running out from the foothills east of the Cascade Crest. The vegetation contrast is moderately visible due to the height of some of the vegetation within this landscape type. The viewer position is level and the view of the right-of-way surface is intermittently screened by the alternating pattern of vegetation and open areas, which helps to screen and absorb the right-of-way vegetation management visual impact. Views of the right-of-way floor can potentially be obtained from the adjacent hillsides and ridgelines. Such views depend upon the horizontal alignment relationship between the right-of-way and the viewer. If the right-of-way runs parallel to the ridgeline or valley wall, the intervening vegetation can potentially screen views of the right-of-way.

6. Valley Floor/Closed Forest -- This zone is typified by valleys approaching the major passes of the Cascade Mountains. The right-of-way vegetation contrast is distinct, due to the difference between the right-of-way and the surrounding landscape. The viewer position is level and with the exception of right-of-way crossings, views are screened by dense vegetation. The right-of-way is potentially visible from adjacent hillsides or ridgelines. The extent of the views obtained from these positions depends upon the horizontal alignment relationship between right-of-way and the viewer position. If the right-of-way is parallel to the valley wall or ridgeline, intervening vegetation will potentially screen views of the right-of-way.

7. Hillside/Grassland-Shrubland -- This zone is typified by the dry, major east-west trending valleys in eastern Washington and Montana. The viewer position is generally below the right-of-way, and thus the right-of-way floor is visible as it proceeds up the enclosing hillsides. The right-of-way is virtually indistinguishable from the surroundings, since the surrounding vegetation carries through the right-of-way without interruption.

8. Hillside/Open Forest -- This zone is typified by areas in the eastern Cascades and the dry exposures of the Northern Rockies. The viewer position is generally below the right-of-way and thus the right-of-way floor is only moderately visible from the surroundings due to the pattern of vegetation clumps and natural clearings. Stands of vegetation provide intermittent screening of the right-of-way.

9. Hillside/Closed Forests -- This category is common in western Washington and Oregon. Viewer position is generally below the right-of-way and the views of the right-of-way floor are obtained as it proceeds up the enclosing hillsides. The right-of-way is clearly visible due to the striking contrast between the open right-of-way and the surrounding masses of dense canopy vegetation. Depending upon the view angle, the dense stands of vegetation can screen views of the right-of-way.

10. Ridge/Grassland-Shrubland -- This zone is typified by major east-west trending valleys in eastern Washington and Montana. The viewer position is generally below the right-of-way at the point where the rights-of-way crosses the ridge. The right-of-way is indistinguishable from the surroundings, since the surrounding vegetation carries through the right-of-way without interruption.

11. Ridge/Open Forest -- This zone is typified by the eastern Cascades and the dry exposures of the Northern Rockies. The viewer position is generally below the right-of-way at the point where the right-of-way crosses the ridge. The right-of-way is virtually indistinguishable from the surroundings due to the pattern of vegetation stands and natural clearings.

12. Ridge/Closed Forest -- This zone is typified by the dense vegetation found in western Washington and Oregon. The viewer position is below the right-of-way at the point where the right-of-way crosses the ridge. The right-of-way is visible because of the notch created in the dense ridgeline vegetation. This notch is probably the most distinct visible feature of the right-of-way vegetation.

Appendices

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