HERBICIDE BACKGROUND INFORMATION

VEGETATIVE MANAGEMENT ENVIRONMENTAL STATEMENT

Pacific Northwest Region
U.S. Department of Agriculture - Forest Service
1974-1975

REPORT

ON

BACKGROUND INFORMATION

FOR

AMITROLE

COMMITTEE MEMBERS:

D. A. Graham R-6

R. Romancier PNW

Peter Thiesen R-6

BACKGROUND DOCUMENT ON AMITROLE

I. General information

References:

(1) Amchem Products, Inc.
1971. Memorandum on amitrole registration. 28 July 1971 (attached).

(2) _____
1972. Selected labels for Amchem Products, Inc. herbicides containing amitrole (attached).

(3) Oregon Extension Service.
1970. Oregon weed control handbook. Oregon State Univ. Coop. Ext. Serv., Corvallis, Oregon. 287 pp.

(4) Washington State University and Department of Agriculture.
1971. Washington pest control handbook. Washington State Univ., Pullman, Washington. 569 pp.

(5) Weed Society of America.
1967. Herbicide handbook of the Weed Society of America. W. F. Humphrey Press, Inc., Geneva, N. Y. 293 pp.

A. Common name: amitrole (5:10)

B. Chemical name: 3-amino-1,2,4-triazole (5:10)

C. Registered uses: Registered for use on annual and perennial grasses and broadleaf weeds, poison ivy, poison oak, and eight species of woody plants (2). Amitrole is registered for use on industrial and other non-crop land (including forest land and rights-of-way) only (1).

D. Formulations manufactured

1. 50% active water-soluble powder: Amchem Weedazol,

2. 90% active water-soluble powder: Amchem Amizol and American Cyanamid Amino Triazole Weed Killer,

3. amitrole + ammonium thiocyanate liquid: Amchem Amitrol-T and American Cyanamid Cytrol Amitrol-T,

4. amitrole + simazine: Amchem Amizine (wettable powder) and Amchem Liquid Amizine (liquid)

E. Dilutions of formulations for use: 5 to 15 gal water per acre for aerial application and 20 to 300 gal water per acre for ground application.

F. Rate and method of application

1. amitrole (Amizol and Amino Triazole Weed Killer): 2 to 10 lb ai/A ground application (2)

2. amitrole + ammonium thiocyanate (Amitrol-T and Cytrol Amitrol-T):
1- to 2-gal (1 gal contains 2 lb each amitrole and ammonium thiocyanate) in 20- to 100-gal water per acre for ground application (2)
½- to 10-gal in 5- to 15-gal water per acre for aerial application (usual aerial spray rate to release conifers from salmonberry in Pacific Northwest is 1 gal amitrole-T in 9 gal water per acre) (2).

G. Tolerances in food or feed and other safety limitations: FDA has declined to set a tolerance for amitrole under terms of FIFRA. Amitrole is an antithyroid agent (goitrogen) and produced thyroid tumors in rats fed at 100 ppm for 68 weeks (1). Amitrole is nonvolatile and nonflammable (5). It is mildly corrosive to bare iron, aluminum, copper, and copper alloys (5). Equipment should be flushed thoroughly with water after use (4, 5).

H. Manufacturer or producer

Amchem Products, Inc.
Ambler, Pennsylvania 19002

American Cyanamid Corporation
P. O. Box 400
Princeton, New Jersey 08540

II. Toxicity data on formulation to be used

References:

(1) Amchem Products, Inc.
1959. Progress report on Amchem Amitrol-T. Amchem Products, Inc. Tech. Data Sheet H-78. 7 pp. mimeo.

(2) Bond, C. E., R. H. Lewis, and J. L. Fryer.
1959. Toxicity of various herbicidal materials to fishes. In Biological problems in water pollution, pp. 96-101. Trans. 1959 Seminar. U.S.H.E.W.

(3) Carter, Mason C.
1969. Amitrole. In Degradation of herbicides (P. C. Kearney and D. D. Kaufman, ed.), pp. 187-206. Marcel Dekker, Inc., N.Y.

(4) Dunachie, J. F. and W. W. Fletcher.
1970. The toxicity of certain herbicides to hen's eggs assessed by the egg-injection technique. Ann. Appl. Biol. 66(3):515-520.

(5) Marston, R. B., D. W. Schults, T. Shiroyama, and L. V. Snyder.
1968. Amitrole concentrations in creek waters downstream from an aerially sprayed watershed sub-basin. Pest. Monit. J. 2:123-128.

(6) Oregon Extension Service.
1970. Oregon weed control handbook. Oregon State Univ. Coop. Ext. Serv., Corvallis, Oregon. 287 pp.

(7) Washington State University and Department of Agriculture.
1971. Washington pest control handbook. Wash. State Univ., Pullman, Washington. 569 pp.

(8) Weed Society of America.
1967. Herbicide handbook of the Weed Society of America. W. F. Humphrey Press, Inc., Geneva, N.Y. 293 pp.

(9) Weir, R. J., O. E. Paynter, and J. R. Elsea.
1958. Toxicology of 3-amino-1,2,4-triazole. Hormolog 2(1):13-14.

Additional references:

general

American Cyanamid Company.
1956. Aminotriazole-acute and subacute toxicity. American Cyanamid Company, Central Medical Dept.

wildlife

Dewitt, J. B., W. H. Stickel, and P. F. Springer.
1963. Wildlife studies, Patuxent Wildlife Research Center. USDI Fish and Wildlife Serv. Circ. 167:74-96.
Includes toxicity of amitrole to bobwhite quail, ring-necked pheasants, and mallard ducks.

aquatic life

Bond, C. E.
1960. Weed control in fish ponds. Oregon Weed Conf. Proc. 9:29-32.

Hughes, J. S. and J. T. Davis.
1962. Toxicity of selected herbicides to bluegill fish. La. Acad. Sci. Proc. 25:86-93.

Lhoste, J.
1959. Dangers to aquatic fauna in the use of chemical herbicides. Phytoma 105:13-17.

bees

King, C. C.
1960. Effects of feeding herbicides to honey bees (Abstr.). N. Central Weed Contr. Conf. Proc. 17:105.

A. Safety data

1. Acute mammalian studies

a. Oral LD_{50}:

	Amitrole (mg/kg)	Amitrole-T (mg/kg)
mice	14,700 (9)	
rats	25,000 (7, 8, 9)	5,000 (6, 7)

Intravenous LD_{50}

mice	1600 no effect (9)
cat	1750 no effect (9)
dog	1200 no effect (9)

b. Dermal LD_{50}: $>$ 10,000 mg/kg (rabbits) for Amizol (7)

c. Inhalation:

d. Eye and skin irritation:

2. Subacute studies

a. Oral: dietary levels of 1000 and 10,000 ppm administered to rats for 63 days resulted in altered body weight gain and fatty metamorphosis of liver cells (9). After 68 weeks of a two-year feeding trial on rats, levels up to 50 ppm have no effect.

At 50 ppm and above, amitrole acts as a goitrogen; the effect is reversible within two weeks after amitrole is withdrawn (9).

b. Dermal

c. Inhalation

Note: poisoning symptoms have not been noted for pure amitrole. In the event of ingestion of amitrole-T, thiocyanate poisoning should be suspected. The acute oral LD_{50} of NH_4SCN is 750 mg/kg (rats) (8).

3. Other studies which may be required

a. Neurotoxicity

b. Teratogenicity: no teratogenic effects in hen's eggs (4)

c. Effects on reproduction

d. Synergism: Experimental results indicate that addition of ammonium thiocyanate to amitrole (amitrole-T) increases degree of control of quackgrass, Bermuda grass, and stoloniferous bent grasses (1). The effect of NH_4SCN is synergistic with the rate of NH_4SCN being more important than the rate of amitrole. Also see:

Boyd, P. G. 1965. Field observations with thiocyanate activated amitrole. Pesticide Progr. 3(6):139.

As noted earlier, the addition of NH_4SCN reduces the LD_{50} value over that of amitrole alone (25,000 mg/kg for amitrole and 5,000 mg/kg for amitrole-T on rats).

e. Potentiation

f. Metabolism

(1) in plants: Amitrole may combine with serine in plants to form 3-(3-amino-1,2,4-triazole-1-yl)-2-aminopropionic acid (3-ATAL) (3). The formation of 3-ATAL apparently represents detoxification, since the derivative is less toxic and less mobile than amitrole. Ammonium thiocyanate, which synergizes the action of amitrole, inhibits the formation of 3-ATAL (3).

Two other unidentified metabolites have been found in some plant species. One of these, unknown III, probably is an artifact of the isolation procedure (3). This compound was five to eight times more active than amitrole on tomato and lettuce roots.

(2) in animals: refer to--

Fang, S. C., S. Khanna, and A. V. Rao.
1966. Further study on the metabolism of labeled 3-amino-1,2,4-triazole and its plant metabolites in rats. J. Agric. Food Chem. 14(3):262-265.

g. Avian and fish toxicity: Amitrole was not toxic to largemouth bass up to 1000 ppm in 48 hour median tolerance tests; the LD_{50} for Coho salmon was 325 ppm for a 48 hour exposure (2). An aerial application of 2 lb ai per acre of amitrole near Astoria, Oregon resulted in such low levels of amitrole that toxicity to warm-blooded animals was unlikely (5). Sampled streams were not buffered against direct herbicide application in this study.

h. Carcinogenicity: Amitrole is an antithyroid agent and has been tested for controlling hyperthyroidism. The stimulation of abnormal growth of the thyroid gland after feeding high dosages of amitrole has been construed as evidence of carcinogenicity. In chronic feeding studies involving exaggerated rates fed over a long period of time, thyroid tumors began appearing in rats fed at 100 ppm for 68 weeks.

B. Physical-chemical properties

References:

( ) Bailey, G. W. and J. L. White.
1965. Herbicides: a compilation of their physical, chemical, and biological properties. Residue Rev. 10:97-122.

(2) Weed Society of America.
1967. Herbicide handbook of the Weed Society of America. W. F. Humphrey Press, Inc. Geneva, N.Y. 293 pp.

1. Boiling point: see (1), melting point 159°C (1, 2)

2. Flash point: nonflammable (2)

3. Physical state: white crystalline powder (2)

4. Density: see (1), molecular weight 84.1 (2)

5. Vapor pressure: nonvolatile

6. Solubility: (2)

Solvent	Temperature (°C)	Solubility (g/100 g)
Acetone	---	Insoluble
Diesel oil	---	Insoluble
Ethanol	75°	26
Ether	---	Insoluble
Kerosene	---	Insoluble
Water	25°	28

7. Stability: stable; no shelf life limitations (2).

III. Efficacy data under field and laboratory conditions

References for effectiveness and phytotoxicity (parts A and B):

(1) Amchem Products, Inc.
1960. Amitrol, b nzac and combinations of both for control of woody plants. Amchem Products, Inc. Tech. Serv. Data Sheet H-79. 5 pp. mimeo. (page 2 attached).

(2) Fechtig, A. D. and W. R. Furtick.
1964. Control of giant Himalaya blackberry (Rubus procerus P. J. Muell) with organic chemical compounds. West. Weed Contr. Conf. Res. Prog. Rpt. 1964:40.

(3) Finnis, J. M.
1964. Chemical control of salmonberry. West. Weed Contr. Conf. Res. Prog. Rpt. 1964:48.

(4) Krygier, James T. and Robert H. Ruth.
1961. Effect of herbicides on salmonberry and on Sitka spruce and western hemlock seedlings. Weeds 9(3):416-422.

(5) Leonard, O. A. and W. A. Harvey.
1965. Chemical control of woody plants. California Agric. Exp. Sta. Bull. 812. 26 pp.

(6) Newton, Michael.
1963. Some herbicide effects on potted Douglas-fir and ponderosa pine seedlings. J. Forestry 61(9):674-676.

(7) Newton, Michael.
1970. Herbicides in forestry. In Oregon weed control handbook. pp. 222-231. Oregon State Univ. Coop. Ext. Serv., Corvallis, Oregon.

(8) Warren, Rex.
1970. Control of common weeds. In Oregon weed control handbook. pp. 247-265. Oregon State Univ. Coop. Ext. Serv., Corvallis, Oregon.

(9) _____
1970. Industrial weed control. In Oregon weed control handbook. pp. 243-244. Oregon State Univ. Coop. Ext. Serv., Corvallis, Oregon.

(10) _____
1970. Weed and brush control along highways, roadways and fence lines. In Oregon weed control handbook. pp. 239-242. Oregon State Univ. Coop. Ext. Serv., Corvallis, Oregon.

(11) _____
1970. Weed control along irrigation and drainage canals. In Oregon weed control handbook. pp. 245-246. Oregon State Univ. Coop. Ext. Serv., Corvallis, Oregon.

A. Effectiveness for intended purpose when used as directed (see table on page 9)

B. Phytotoxicity (see table on page 9)

RELATIVE EFFECTIVENESS OF AMITROLE AND AMITROLE-T
APPLIED AS FOLIAGE SPRAYS
FOR SPECIFIC SPECIES AND WEED CONTROL PROBLEMS

Weed Control Problem	Species	Application Method	Herbicidal Combination	Results	References
Woody plants non-crop lands	ash, white[1/]	spot		good	1
	blackberry[1/]	spot		good	1, 2, 5, 7
	cascara	spot, aerial		good	5, 7
	cherry, black[1/]	spot		good	1
	Douglas-fir	spot, aerial		damage	6
	elder, red	spot		good	7
	hemlock, western	spot, aerial		damage	4
	honeysuckle, Japanese[1/]	spot		good	1
	ivy, poison[1/]	spot		good	1
	kudzuvine[1/]	spot		good	1
	locust, black[1/]	spot		good	1
	maple, bigleaf[1/]	spot, aerial		fair	7
	oak, California black	spot		good	7
	oak, Oregon white	spot		good	7
	poison oak[1/]	spot, aerial		good, fair	1, 5, 7, 8
	salmonberry[1/]	spot, aerial		good	1, 3, 4, 5, 7
	spruce, Sitka	spot, aerial		resistant	4
	sumac, staghorn[1/]	spot		good	1
Herbaceous plants--non-crop lands	cattail[1/]	broadcast, aerial		good	8
	Canada thistle[1/]	spot		good	8
	horsetail rush[1/]	spot		good	8
	quackgrass[1/]	spot		good	8
	white top[1/]	spot		good	8
Herbaceous plants--rights-of-way and industrial sites	annual grasses[1/] and broadleaf weeds	broadcast	+ atrazine, bromacil, or Tandex	good	9, 10
Herbaceous plants--irrigation or drainage canals	general weed[1/] control without sterilization		+ 2,4-D	good	11

[1/] Represents registered use

C. Translocation in plant treated

References:

(1) Clor, M. A., A. S. Crafts, S. Yamaguchi.
1964. Translocation of C^{14}-labeled compounds in cotton and oaks. Weeds 12(3):194-200.

(2) Crafts, A. S.
1961. The chemistry and mode of action of herbicides. Interscience, N.Y. 269 pp.

(3) Forde, B. J.
1966. Translocation patterns of amitrole and ammonium thiocyanate in quackgrass. Weeds 14(2):178-179.

(4) Leonard, O. A.
1963. Translocation of herbicides in woody plants. Soc. Amer. Foresters Proc.

(5) Leonard, O. A., D. E. Bayer, and R. K. Glenn.
1966. Translocation of herbicides and assimilates in red maple and white ash. Bot. Gazette 127(4):193-201.

Amitrole applied to either leaves or stems was absorbed and transported throughout red maple and white ash trees (5). Amitrole apparently moves both in the cell wall (apoplast) and living protoplasm (symplast) of plants (4). Applications to leaves move downward and throughout the plant; applications to lower stems or roots apparently move upward in the transpiration stream (2). Translocation of amitrole from the leaf of quackgrass was retarded over 12 hours by ammonium thiocyanate applied as a spray or as a spot (3). When amitrole was applied without NH_4SCN, there was considerable movement to immature leaves and roots. After 24 hours, marked symplastic movement occurred; plants treated with both chemicals showed the most movement (3).

D. Persistence in soil, water, or plants

References:

(1) Carter, Mason.
1969. Amitrole. In Degradation of herbicides. pp. 187-206. Marcel Dekker, N.Y.

(2) Day, B. E., L. S. Jordon, and R. T. Hendrixson.
1961. The decomposition of amitrole in California soils. Weeds 9(3):443-456.

(3) Frear, D. E. H.
1964. Fate of 3-amino-1,2,4-triazole in soils. J. Sci. Food Agric. 15(8):II-85-5.

(4) Freed, V. H. and W. R. Furtick.
1961. The persistence of amitrole in soil when used for chemical fallow. Hormolog 3(1).

(5) Ludzack, F. J. and J. W. Mandia.
1962. Behavior of amitrole in surface water and sewage treatment. Proc. 16th Ind. Waste Conf., Purdue Univ. Engng. Ext. Serv. No. 109:540.

(6) Marston, Richard B., Donald W. Schults, Tamotsu Shiroyama, and Larry V. Snyder.
1968. Pesticides in water: amitrole concentrations in creek waters downstream from an aerially sprayed watershed sub-basin. Pest. Mont. J. 2(3):123-128.

(7) Norris, Logan A.
1967. Chemical brush control and herbicide residues in the forest environment. In Herbicides and vegetation management. pp. 103-123. School of Forestry, Oregon State Univ., Corvallis, Oregon.

(8) _____
1970. Degradation of herbicides in the forest floor. In Tree growth and forest soils (Youngberg, C. T. and C. B. Davey, ed.) pp. 397-411. Oregon State Univ. Press, Corvallis, Oregon.

(9) _____
1970. The kinetics of adsorption and desorption of 2,4-D, 2,4,5-T, picloram, and amitrole on forest floor material. West. Soc. Weed Sci. Res. Prog. Rpt. 1970:103-105.

(10) Norris, L. A., M. Newton, and J. Zavitkavoski.
1966. Stream contamination with amitrole following brush control operations with amitrole-T. West. Weed Contr. Conf. Res. Prog. Rpt. 1966:20-22.

(11) Norris, L. A., M. Newton, and J. Zavitkavoski.
1967. Stream contamination with amitrole from forest spray operations. West. Weed Contr. Conf. Res. Prog. Rpt. 1967:33-35.

(12) Sund, Kenneth A.
1956. Residual activity of 3-amino-1,2,4-triazole in soils. Agric. Food Chem. 4(1):57-60.

1. Soil:

Amitrole residues could not be detected two months after application of one- to two-pounds per acre on three soil types in Oregon (4). Amitrole was adsorbed in red alder humus more rapidly than it was desorbed (9). After 35 days, recovery of amitrole from red alder floor material had dropped to 20 percent (8). The presence of 2,4-D or ammonium thiocyanate are not likely to influence the persistence of amitrole in the field (8:407). Degradation of amitrole proceeded at a near normal rate in steam-sterilized forest floor material despite nearly complete absence of biological activity (8:408). Amitrole appears to become tightly adsorbed to soil particles and can complex metals (12). It may also act in the soil's base exchange system (11). Amitrole disappears rapidly from soils. Disappearance has been attributed to adsorption, microbial degradation, and nonbiological destruction (1). Evidence indicates that nonbiological destruction is the most important cause of amitrole disappearance in soils (1).

2. Water:

Amitrole was not degraded by biologic action in river water, sewage, activated sludge, or anaerobic digestion tests (5). Amitrole interfered with nitrification in river water and activated sludge. Clorination degraded amitrole to unidentified compounds. Studies of amitrole contamination in streams following aerial applications indicate that maximum residues occur immediately after spraying and decline rapidly (6, 7, 10, 11). Maximum concentration of 155 ppb at the downstream edge of a 100-acre unit treated at two pounds per acre was attained 30 minutes after application began (6). It decreased to 26 ppb by the end of the two hour application and to non-detectable amounts six days after spraying. No amitrole was detected at any time 1.8 miles below the sprayed area. In another study, maximum concentration immediately downstream from the sprayed area was 422 ppb

0.17 hours after spraying and dropped to 6 ppb 8 hours after spraying (7). Residues did not persist into the next year and heavy rains six months after application did not introduce measurable amounts of amitrole into the same stream (11).

3. Plants:

The *s*-triazole nucleus is highly stable and few workers have reported evidence of ring cleavage under physiological conditions (1). The half-life of amitrole in corn was about 8 days. Disappearance in soybeans was much slower. Amitrole could not be detected in cotton after 4 days but large quantities of metabolic products were present. Ring cleavage has been observed in oats and barley but not in beans and tomatoes. Photodecomposition in the presence of riboflavin has also been reported. Amitrole degradation in plants seems to involve conjugation between amitrole and endogenous plant constituents. These products contain the intact triazole nucleus which often can be regenerated by chemical treatment. The principal detoxification product seems to be 3-(3-amino-1,2,4-triazole-1-yl)-2-aminopropionic acid (3-ATAL).

E. Compatibility with other chemicals

Amitrole and amitrole-T are compatible with many other herbicides, but other pesticides and fertilizers should be used with caution. All amitrole formulations can be mixed with 2,4-D, 2,4,5-T, simazine (and other *s*-triazines), bromacil, and diuron.

IV. Environmental impact

A. Effects of pesticide on non-target organisms.

B. Residues in or on food or feed.

Reference: (1) Ecological effects of pesticides on non-target species. 1971. 220 pp. U.S. Government Printing Office S/N 4106 - 0029. (Listed in Selected U.S. Govern. Publ. 1(3)--1972).

The U.S. Government publication (1) contains pertinent information for each pesticide discussed concerning non-target mammals, birds, fishes, amphibians, mollusks, arthropods, annellids, plants, and microorganisms. It also presents information on biological concentration in food chains and persistence for each pesticide.

(2) Norris, Logan A.
1971. Chemical brush control: assessing the hazard. J. Forestry 69(10):715-720.

Logan Norris (2) concludes that the relatively large doses of amitrole required to produce acutely toxic responses in most non-target organisms are not likely to occur from normal chemical brush control operations on forest lands. The short persistence, lack of biomagnification in food chains, and the rapid excretion by animals preclude chronic exposure and, therefore, chronic toxicity.

REPORT

ON

BACKGROUND INFORMATION

FOR

ATRAZINE

Assignment:

Pesticide: Atrazine (AAtrex)

Use: Herbicide

Priority: 1

Team: M. Weiss (R-3) (For F. Yasinski (R-3))
W. Davis (R-4)
H. Pangman (R-4)

I. GENERAL INFORMATION

A. Common Name. Atrazine[1/]

B. Chemical Name. 2-chloro-4-ethylamino-6-isopropylamino-s-triazine (Anon., 1971a)

C. Registered Uses. AAtrex 80W is registered for season-long weed control in corn and sorghum and for weed control in certain other crops; in non-crop areas; and industrial sites (Anon., 1971b).[2/] AAtrex 80W is registered for use in forest and Christmas tree plantations of Douglas-fir, grand fir, noble fir, white fir, lodgepole pine, ponderosa pine, and Scotch pine (The registration for AAtrex 80W limits its use in forest and Christmas tree plantations to the Pacific Northwest, west of the Cascades.) (Anon., 1971b)

D. Formulations Manufactured. AAtrex 80W, a wettable powder containing 80% active ingredient (Anon., 1971a). [2/]

E. Dilution of Formulation to Use.

1. Forest and Christmas Tree Plantations (Anon., 1971b)

For annual broadleaf and grass weed control - 2½-5 pounds of AAtrex 80W is diluted in 20-40 gallons of water per acre.

For quackgrass control - 5 pounds of AAtrex 80W is diluted in 20-40 gallons of water per acre.

2. Nonselective Weed Control on Non-Crop Land (Anon., 1971b)

Use sufficient water to assure thorough coverage. Use at least 1 gallon of water for each pound of AAtrex 80W; more if practical.

F. Tolerances in Food or Feed and Other Safety Limitations

1. Tolerances for Residues of Atrazine (Anon., 1971b)

Tolerances for residues of atrazine on certain raw agricultural commodities have been set as follows:

15.00 ppm In or on corn forage or fodder (including field corn, sweet corn and popcorn), perennial ryegrass, sorghum fodder and forage.

1/ Trademark: AAtrex, Gesaprim
2/ AAtrex 4L, a liquified formulation containing 4 lbs. of technical atrazine per gallon, is registered for season-long weed control in corn and sorghum (Anon., 1971c)

10.00 ppm In or on pineapple fodder and forage.

5.00 ppm In or on wheat fodder and straw.

0.25 ppm In or on fresh corn including sweet corn (kernels plus cobs with husks removed) corn grain (includes popcorn), macadamia nuts, pineapples, sorghum grain, sugarcane, sugarcane fodder and forage, wheat grain.

0.02 ppm In eggs, milk, meat, fat and meat by-products of cattle, goats, hogs, horses, poultry and sheep.

2. Other Safety Limitations

a. "Care should be taken to avoid using AAtrex where adjacent desirable trees, shrubs, or plants might be injured" (Anon.,1971b).

b. Forest and Christmas Tree Plantations (Anon., 1971b)

"Do not graze treated areas. Do not apply to seedbeds. Do not make more than one application per year."

c. Nonselective Weed Control on Non-Crop Land.
(See Supplemental page 1)

G. Rate and Method of Application

1. Forest and Christmas Tree Plantations (Anon., 1971b).

For annual broadleaf and grass weed control, AAtrex is applied broadcast at rates of 2 to 4 pounds active ingredient per acre. It is applied "between fall and early spring while trees are dormant or soon after transplanting and before weeds are 1½ inches high." For band application, the rate is reduced in proportion to the area treated.

For quackgrass control, AAtrex is applied broadcast at rates of 4 pounds active ingredient per acre. It is applied "in fall or early spring while trees are dormant and before weed seedlings are more than 1½ inches high."

2. Nonselective Weed Control on Non-Crop Land (Anon., 1971b)

AAtrex 80W is applied "before or soon after weeds begin growth."

To control most annual broadleaf and grass weeds (such as barnyardgrass, cheatgrass, crabgrass, lambsquarters, foxtail, ragweed,

puncturevine, and turkey mullein), AAtrex 80W is applied broadcast at rate of 4.8 to 10 pounds active ingredient per acre.

To control hard-to-kill annual and many perennial broadleaf and grass weeds (such as bluegrass, burdock, Canada thistle, dogfennel, orchardgrass, plantain, quackgrass, purple top, redtop, and smooth brome), AAtrex 80W is applied broadcast at rates of 10 to 20 pounds active ingredient per acre.

To control hard-to-kill biennial and perennial weeds (such as bull thistle and sowthistle), AAtrex 80W is applied broadcast at rates of 20 to 40 pounds active ingredient per acre.

For longer residual control in regions of high rainfall and a long growing season, AAtrex 80W is applied broadcast at rates of 20 to 40 pounds active ingredient per acre.

3. "In each case where a range of rates is given, the lower rate should be used on light soils and soils low in organic matter; the higher rate should be used on heavy soils and soils high in organic matter" (Anon., 1971b)

H. Manufacturer or Producer

Geigy Agricultural Chemicals
Division of CIBA-GEIGY Corporation
Ardsley, New York 10502

II. TOXICITY DATA ON FORMULATION TO BE USED

A. Safety Data

1. Acute Mammalian Studies

a. Oral

Species	Formulation	Dosage LD_{50}
Albino rats	Technical	3,080 mg/kg (Anon., 1971a)
Albino mice	Technical	1,750 mg/kg (Anon., 1971a)
Albino rats	80W	5.1 $\pm$ 0.4 g/kg (Anon., 1971a)

Palmer and Radeleff (1969) studied the toxicity of atrazine and other herbicides to cattle, sheep, and chickens. For atrazine,"Cattle and sheep were dosed by either drench or capsule, chickens by capsule. The toxic dosage for cattle was 25 mg/kg after 8 doses by drench and 2 by capsule. The toxic dosage for sheep was 5 mg/kg." "However, one sheep received 199 consecutive doses at 50 mg/kg before it was poisoned and died. Chickens

given 10 at 50 mg/kg had a significant reduction in weight gains." (See Supplemental pages 1 and 2)

b. Dermal

Species	Formulation	Dosage LD_{50}
Albino rabbits	80W	9.3 ± 0.9 g/kg (Anon., 1971a)

c. Inhalation. "there has been no evidence of toxicity in rats subjected to aerosol dust containing the equivalent of 1.6 mg/liter of technical grade atrazine." (Anon., 1971a)

d. Eye and Skin Irritation

2. Subacute Studies

a. Oral

"No observable ill effects have been detected in cattle, dogs, horses, or rats fed a diet which included more than 25 ppm atrazine over extended periods." (Anon., 1971a)

"Administration of daily dosages of 100 ppm of an 80% wettable powder formulation of atrazine to cows for 21 days or feeding 30 ppm of this formulation in grain to cattle for four weeks resulted in no observable effect." (Anon., 1971a)

3. Other Studies Which May Be Required

a. Teratogenicity and Carcinogenicity. "Long term studies in rats and mice have revealed no carcinogenic or teratogenic effects either in the parents or progeny following long term administration of atrazine." (Anon., 1971a)

The Technical Panel on Carcinogenesis of the Secretary's Commission on Pesticides and Their Relationship to Environmental Health (Anon., 1969) examined the available reports on tests of tumorigenicity conducted on about 100 pesticidal chemicals and assigned each of the pesticides to one of four groups: A, B, C, or D. Atrazine was placed in the group containing those pesticides for which the available evidence was considered insufficient for judgement (Group C). Atrazine was further placed in Priority Group C4, one of four priority groups in Group C. Priority Group C4 was characterized by "Tumor incidence not elevated in adequate studies conducted in one species [mouse] only but current guidelines require negative results in two animal species for judgements of negativity."

Chapter 8 of the Report of the Secretary's Commission on

Pesticides and Their Relationship to Environmental Health (Anon. 1969) contains information on tests run by the Bionetics Research Laboratories of Litton Industries with various pesticides and related compounds for teratogenic effects. "The Bionetics data were reanalyzed statistically to account for litter effects." The data for atrazine was placed in Table 3, the table containing data on "Tests which showed no significant increase of anomalies (with particular doses, solvents, or test strains)." The data for atrazine from table 3 was as follows:

Compound	Strains	Solvent	Dose per kg. body wt.	Increased mortality (C57BL/6)	Total number of litters
Atrazine	C3H	DMSO	46.4 mg.	-	6
Do	C57	DMSO	46.4 mg.	-	13
Do	AKR	DMSO	46.4 mg.	-	15

b. Mutagenicity

Table 3, page 584, of the Report of the Secretary's Commission on Pesticides and Their Relationship to Environmental Health (Anon., 1969) contains a "List of various pesticides (1000 ppm, 12 hrs.) known to produce mutations in barley and relative efficiency of each to control and to 5,500 R of X rays (Wuu and Grant, 111)."[1/] Atrazine is listed as having a relative efficiency of 10, X rays a relative efficiency of 32, and control a relative efficiency of 1.

Page 639 of the Report of the Secretary's Commission on Pesticides and Their Relationship to Environmental Health (Anon., 1969) contains data collated from files of the Environmental Mutagen Information Center. Data on atrazine was presented as follows:

Pesticide	Organism in which tested	Assay system	Dose	Biological effect	EMIC registry No.
Atrazine	Barley	Anther	1000 ppm-Soaked	Slight effect on meiosis (C_1)	70 2/
				Slight effect on meiosis (C_2)	70 2/

1/ The paper cited (Wuu and Grant,111) was Wuu, K. D. and W. F. Grant. Morphological and somatic chromosomal aberrations induced by pesticides in barley (Hordium vulgare). Can. J. Genet. cytol. 8: 481-501, 1966

2/ EMIC registry No. 70 is for the following paper: Wuu, K. D. and W. F. Grant. Chromosomal Aberrations Induced by Pesticides in Meiotic Cells of Barley. Cytologia 32: 31-41., 1967.

c. Avian and Fish Toxicity

Species	Formulation	Route Administered	Dosage LD_{50}-LC_{50} (Anon, 1971a)
Mallard duck	Technical	5-day feeding	19,560 ppm (Anon., 1971a)
Bobwhite quail	Technical	7-day feeding	5,760 ppm (Anon., 1971a)
Rainbow trout	Technical	96-hr. exposure	4.5 ppm (Anon., 1971a)
Bluegill sunfish	Technical	96-hr. exposure	24 ppm (Anon., 1971a)
Goldfish	Technical	96-hr. exposure	60 ppm (Anon., 1971a)

Species	Formulation	Sex	Age	LD_{50} (95% Conf. lim.) mg/kg	
Mallards	80% Wettable powder	Female	6 mos.	>2000	Tucker and Crabtree (1970)

B. Physical-Chemical Properties

1. Physical State. White, crystalline substance which is non-combustible and non-corrosive. (Anon., 1971a)

2. Vapor Pressure. At 20° C: 3.0×10^{-7} mm of Hg (Anon., 1971a).

3. Solubility at 27° C. (Anon., 1971a)

Solvent	ppm
Water	33
n-Pentane	360
Diethylether	12,000
Methanol	18,000
Ethyl acetate	28,000
Chloroform	52,000
Dimethyl sulfoxide	183,000

4. Stability. (See Supplemental page 3)

"Atrazine is stable in neutral, slightly acid, or basic media. It sublimes at high temperatures and when heated, especially at high temperatures in acid or basic media, hydrolyzes to 2-hydroxy-4-ethylamino-6-isopropylamino-s-triazine which has no herbicidal activity." (Anon., 1971a)

"Shelf life of the formulated 80W product in unopened paper or polyethylene bags is more than five years." (Anon., 1971a)

III. EFFICACY DATA UNDER FIELD AND LABORATORY CONDITIONS

A. Effectiveness for Intended Purpose When Used as Directed

Bickford et al. (1965) found that atrazine improves survival of Douglas-fir seedlings and ponderosa pine seed spots. Atrazine was applied in March at 5, 3 1/3, and 1 2/3 pounds per acre of 80% active material to plots near Corvallis, Oregon. Seedlings were planted on five dates from November to March and spots were seeded in April. Survival of seedlings was "doubled on the plots where 1 2/3 pounds an acre was applied, and increased to nearly five times the survival obtained on untreated plots, in situations where 3 1/3 pounds were applied. There was little difference in survival between plots with 3 1/3 pounds an acre and plots with 5 pounds an acre." "Response from seeded plots indicated comparable weed-control requirements for planted Douglas-fir and seeded ponderosa pine."

Newton (1964) tested atrazine; Amitrol, Simazine, at a 1:3 mixture; Simazine; and Isocil for weed control in planted Douglas-fir. On treated plots, half of the seedlings were planted before treatment and half were planted after treatment. Spring treatments with atrazine at 5 pounds active ingredient per acre gave complete weed control with no conifer damage. "Fall treatments produced poorer weed control and possibly some conifer damage." Newton "concluded that spring applications of herbicides are generally superior to fall treatments in this region of low summer precipitation and wet winters, and that it is wise to avoid chemicals which either allow rapid regrowth of weeds, or damage seedlings through their own toxicity. These results suggest atrazine as the most promising chemical for this type of treatment under local conditions, and rates of roughly four pounds active material in spring applications."

Gratkowski (1971) tested atrazine and several other chemicals for grass and forb control in Douglas-fir plantations at four locations in southwestern Oregon. Two of the locations were on the wet coastal slope of the coast range and two were in the dry interior valleys. "Terbacil proved most promising in these tests." In discussing the reasons for his tests, Gratkowski states that "Atrazine is widely used for grass control in plantations, but it is relatively ineffective on broadleaf weeds."

Newton and Webb (1970) in the summary of their paper state that "The limited information available suggests that herbicides applied successfully in regeneration of Douglas-fir may largely be considered effective and safe for ponderosa pine. With atrazine or dalapon, or both, herbaceous weed control should be sufficient to establish pine in most areas. Two, 4-D and other foliage - active compounds may be applied, but with full consideration of the hazards involved. Ponderosa pine is resistant to atrazine, but the threshold of resistance is probably lower in coarse-textured soils. Resistance of weeds to atrazine will be comparably lower in the coarse-textured soils, however, and less material is needed to

accomplish the same job of weed control with the same degree of safety. On some soils, largely rocky and coarse textured or gravelly, vegetation is not responsible for rapid drying. On such sites, good control of weeds alone is not sufficient to guarantee survival."

B. Phytotoxicity (See Supplemental page 4)

"Plant species such as corn and sorghum have the ability to readily metabolize atrazine into nonphytotoxic compounds, therefore, they are resistant to rates of atrazine commonly used for weed control. Other plant species differ in their abilities to metabolize atrazine so various degrees of susceptibility can be seen." (Anon., 1971a)

Kozlowski and Kuntz (1963) studied the effects of atrazine and other herbicides on red pine (Pinus resinosa Ait.) and white pine (Pinus strobus L.) seedlings. They found that applying simazine, atrazine or propazine as a pre-emergence spray or directly to recently germinated seedlings caused severe damage. As a pre-emergence treatment, atrazine was applied immediately after planting at rates of 1, 2, and 4 pounds per acre. Atrazine did not affect seed germination. However, "atrazine adversely affected seedling growth and caused varying degrees of mortality. Within seven days after emergence, a slight needle curling was observed. Chlorosis developed and growth was visibly depressed. Adverse effects increased with time." "One month after germination, approximately 10 percent of the seedlings had died in flats treated with 4 pounds atrazine per acre."

As a post-emergence treatment, atrazine was applied 3 weeks after seeding. "Seed germination and emergence of white pine continued over a 3 week period." Atrazine was sprayed at rates of 1, 2, and 4 pounds per acre. One month after treatment all red pine seedlings were dead on both soil and sand at all rates. "The following percentages of white pine seedlings were killed on treated soil and treated sand, respectively, at the indicated rates (per acre): 4 pounds,82 and 90 percent; 2 pounds, 67 and 91 percent; and 1 pound, 55 and 33 percent." "In general, the seedlings which had emerged prior to treatment suffered somewhat less injury and mortality than did the seedlings which emerged after treatment."

"Simazine, atrazine, and propazine did not leach readily from the surface inch of Plainfield sand when 2, 4, or 8 surface inches of water were applied. Some atrazine, however, moved downward from the first inch more readily than did simazine or propazine." "No injury occurred to 2-0 red pine when simazine was applied to the soil surface at 4 or 8 pounds per acre or when simazine was applied to the foliage only. When, however, amounts of simazine equivalent to 4 or 8 pounds per acre (soil-surface basis)were incorporated into the soil, especially in the root zone, severe injury resulted and seedlings eventually were killed." "These experiments emphasized that whereas young seedlings are killed by triazine herbicides,

older seedlings are not, because the roots of the older seedlings normally are below the layers of soil which contain phytotoxic amounts of these chemicals."

Kozlowski and Torrie (1965) studied the effect of soil incorporation of atrazine and other herbicides on germination and development of very young red pine (Pinus resinosa Ait.) seedlings. Atrazine was sprayed at rates of 2, 4, 8, and 16 pounds per acre to the surface of flats containing Plainfield sand. The following day the soil was mixed and pine seeds were planted. "Soil-incorporated atrazine was exceedingly toxic to the seedlings at all dosages." With the exception of ipazine up to 4 pounds per acre, the soil-incorporated triazine herbicides were generally very toxic to young pine seedlings. "The toxicity varied greatly in the following decreasing order: atrazine, simazine, prometryne, propazine, ipazine." "Certain triazines exhibited greatly delayed toxicity. For example, more seedlings died in the last 20 days of the experiment than in the first 90 days . . ." "Seed germination was influenced only very slightly or not at all, by a variety of soil-incorporated herbicides. In contrast, all but one of the soil-incorporated herbicides caused seedling mortality and decreased dry-weight production of seedlings in varying amounts," "The toxicity of soil-incorporated herbicides was generally much greater than when the same herbicides were applied to the soil surface."

Walker (1964) tested atrazine and other s-triazine compounds as aquatic herbicides. Atrazine was applied in the field in open plots, whole ponds, and to plastic enclosures. Atrazine was applied at rates of 0.2 to 6.0 ppmw to 11 submerged plant species and 4 kinds of filamentous algae. "Eradication was most consistent in water treated in total volume dosages. Weeds were controlled in ponds or plastic enclosures while poor results were obtained in partial treatments of open plots. Atrazine concentrations of 0.5 to 1.0 ppmw effectively controlled most filamentous algae and pondweeds in pond applications. Spray applications of wettable powder generally were more effective than broadcasting granular atrazine. The duration of phytotoxity varied from seasonal control or growth inhibition achieved at the lower concentration (0.5 ppmw) to complete eradication which exceeded a year's length as the result of higher rates (1.0 ppmw). Emergent grasses and herbaceous plants also were affected in a manner similar to simazine applications." "Simazine and atrazine were slow to give results in the aquatic environment. A two- to six-week lapse was required before phytotoxity symptoms were noted. The characteristic herbicidal effect of simazine, atrazine, and propazine was a chlorotic appearance along with progressive decomposition of affected plant parts. Inhibition of plant growth often was accompanied by spotty eradication of rooted aquatic plants and filamentous algae. Phytoplankton tubidity was curtailed temporarily following the herbicide applications. Once the higher plants decayed, zooplankton also became abundant." "The ecological sequence of the secondary succession was noted following the eradication of aquatic vegetation. The use of plastic enclosures allowed critical comparison of the ecological changes

produced by various rates and plant conditions." "Algae rarely were controlled for more than one season. Inhibition of phytoplankton was temporary, lasting less than 2-3 months in most applications."

C. Translocation with Plant Treated

"Atrazine enters plants primarily through the root system. Inside the plant it crosses cortical tissue to the xylem. The xylem appears to be the principal tissue by which atrazine is translocated. Atrazine is translocated upward in plants and upon accumulating in photosynthetic tissues (i.e. chloroplasts), the plants die. Atrazine is also absorbed to some extent through the foliage." (Anon., 1971a)

D. Persistence in Soil, Water, or Plants (See Supplemental page 5)

Kearney (1970) refers to an extensive review of the literature by himself and others[1/] on the persistence of 11 major classes of pesticides in soils. The triazine herbicides were grouped with the urea and picloram herbicides. For the group, the time required for loss of 75 to 100 percent activity is 18 months.

Kozlowski and Kuntz (1963) found that "when Plainfield sand to which 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 used in leaching. However, some herbicide, especially atrazine, moved downward to a 6-inch depth. With increased amount of leaching more herbicide was translocated out of the first inch of atrazine- treated soil. Such an effect was not as apparent with simazine - or propazine - treated soil. The greater leachability of atrazine was probably related to its greater solubility." "This study, which demonstrates the difficulty of removing triazine herbicides from upper soil levels even with large amounts of water, emphasizes the dangers of possible persistence and accumulation of triazine herbicides in forest nurseries, even in light sandy soils."

E. Compatibility with Other Chemicals

"When weeds are resistant to AAtrex, combinations of AAtrex with sodium chlorate formulations, dalapon, TCA, amitrole, simazine (Princep), and other compounds may be used to broaden the spectrum of weed control." (Anon., 1971a)

1/ Kearney, P. C. , R. G. Nash, and A. R. Isensee, 1969. Persistence of pesticide residues in soils, Chapter 3, p.p. 54-67. In M. W. Miller and G. C. Berg (eds.). Chemical fallout: Current research on persistent pesticides, Springfield, Ill. Thomas .

IV. ENVIRONMENT IMPACT

A. Effects of Pesticide on Non-Target Organisms

Walker (1964) studied atrazine and other s-triazine compounds as aquatic herbicides. Atrazine was applied at rates of 0.2 to 6.0 ppmw to 11 submerged species and 4 kinds of filamentous algae. Samples of bottom fauna organisms were taken from plastic enclosures used in the study. "Determination of acute toxicity to organisms was based upon comparison of samples obtained from the treated area and untreated control up to six weeks following the application. Chronic toxicity was measured by comparative production three months to a year following the application." "The herbicidal destruction of plant cover in fish habitat exposes smaller forage fishes to predation by larger sport fishes. No toxicity to fishes was demonstrated by the application of the s-triazine compounds under field conditions." "In contrast to simazine, atrazine was somewhat toxic to bottom fauna. Among the most sensitive organisms were mayflies (Ephemeroptera), caddis flies (Tricoptera), leeches (Hirudinea), and gastropods (Musculium). The most significant reduction in bottom fauna was observed during the period immediately following the application. Bottom fauna appeared to recover according to observations made four to six months following the treatment in the simazine tests."

B. Residues

St. John et al. (1964) studied the fate of atrazine and other chemicals in the dairy cow. "Four holstein cows were catheterized and each was fed one of the herbicides at the 5 ppm level (based on a daily ration of 50 lb.) for four days. The pure herbicides in absolute ethyl alcohol (except atrazine, which was dissolved in acetone) were mixed with the grain. Morning and evening subsamples of the total mixed milk were taken one day prior to feeding (control sample), daily throughout the feeding period, and for two days thereafter. The total daily urine sample was similarly collected, weighed, mixed, and subsampled over the same test period." "A colorimetric method and isolation procedure was developed for atrazine in milk and urine based on the Zincke reaction with active halogen compounds . . ." "The residue determined represented intact atrazine, since the Zincke reaction is applicable only to compounds containing active halogens. Atrazine may have been largely converted to hydroxy atrazine and excreted in the urine as a water-soluble conjugate of this compound." "No residues of these herbicides were found in the milk. About 2% of intact atrazine was eliminated in the urine."

Norris et al. (1967) "made a preliminary survey of atrazine residues in deer harvested from forest lands treated with this herbicide for grass control. Deer were harvested at various intervals after application of the herbicide [17 days, 26 days, and 44 days], and various organs and body tissues were removed, placed in plastic bags, and frozen

as quickly as possible. The analytical procedure was essentially that outlined in Geigy Analytical Bulletin Number 7 with the exception that the herbicide was determined with a gas chromatograph." "Unfortunately, a control animal was not available; so there is no indication whether or not deer normally carry atrazine residues, however this possibly appears quite unlikely. On the basis of our analysis using two different columns and a halogen specific detection system there is little question that the chemical measured is in fact atrazine." "We found no atrazine residue greater than 76 ppb in portions of these animals which might normally be used for human consumption. In one animal, not listed above, residues of 326 ppb atrazine were found in the thyroid and 498 ppb in the lymph glands. Another animal yielded a fat sample which contained 688 ppb atrazine."

"In general this survey indicates that atrazine applied for grass control on forest lands of southern Oregon will enter several tissues and organs of deer. The length of persistence of the chemical in these tissues is not clear from this study. The likelyhood (sic.) of encountering dangerous residues of atrazine in tissues of importance for human consumption appears low."

CITED REFERENCES

Anon. 1969. Report of the Secretary's Commission on Pesticides and Their Relationship to Environmental Health, Parts I and II, U. S. Department of Health, Education and Welfare. 677 p.

Anon. 1971a. AAtrex herbicide technical bulletin. Geigy Agricultural Chemicals. GAC 700-564. 8 p.

Anon. 1971b. AAtrex 80W herbicide sample label. Geigy Agricultural Chemicals. GAC 130-069. 8 p.

Anon. 1971c. AAtrex 4L herbicide sample label. Geigy Agricultural Chemicals. GAC 130-070. 4 p.

Bickford, M. L., J. Zavitkovski, and M. Newton, 1965. Atrazine improves survival of Douglas-fir seedlings and ponderosa pine seed spots. Research Progress Report. Research Committee Western Weed Control Conference. 48-49.

Gratkowski, H. 1971. Grass and forb control in Douglas-fir plantations. Research Progress Report. Research Committee Western Society of Weed Science. 31.

Kearney, P. C. 1970. Summary and conclusions. in Residue Reviews. Vol. 32. Single Pesticide Volume: The Triazine Herbicides. 391-399.

Kozlowski, T. T. and J. E. Kuntz. 1963. Effects of simazine, atrazine, propazine, and eptam on pine seedlings. Soil Sci. 95: 164-174.

Kozlowski, T. T. and J. H. Torrie. 1965. Effect of soil incorporation of herbicides on seed germination and growth of pine seedlings. Soil Sci. 100(2): 139-146.

Newton, Michael. 1964. Chemical weed control in conifer plantations. Research Progress Report. Research Committee Western Weed Control Conference. 42-43.

Newton, Michael, and W. L. Webb. 1970. Herbicides and the management of young pine. Symposium Proceedings: Regeneration of Ponderosa Pine. School of Forestry. Oregon State University, Corvallis. 94-99.

Norris, Logan A., M. Newton, and J. Zavitkovski. 1967. Atrazine residues in deer. Research Progress Report. Research Committee Western Weed Control Conference. 30-31.

Palmer, J. S., and R. D. Radeleff. 1969. The toxicity of some organic herbicides to cattle, sheep, and chickens. U. S. D. A. A. R. S. Production Research Report No. 106. 26 p.

St. John, L. E., D. G. Wagner, and D. J. Lish. 1964. Fate of atrazine kuron, silvex, and 2,4,5-T in the dairy cow. J. Dairy Sci. 47(11): 1267-1270.

Tucker, R. K. and D. Glen Crabtree. 1970. Handbook of Toxicity of Pesticides to Wildlife. U. S. D. I. Fish and Wildlife Service. Bureau of Sport Fisheries and Wildlife. Resource Publication No. 84. 131 p.

Walker, Charles R. 1964. Simazine and other s-triazine compounds as aquatic herbicides in fish habitats. Weeds 12(2): 134-139.

Additional Information

I. GENERAL INFORMATION

F. Tolerances in Food or Feed and Other Safety Limitations

2. Other Safety Limitations

c. Nonselective Weed Control on Non-Crop Land

"Do not contaminate domestic or irrigation water supplies, or lakes, streams or ponds." (Anon., 1971. AAtrex 80W herbicide sample label. Geigy Agricultural Chemicals. GAC 130-069. 8 p.)

II. TOXICITY DATA ON FORMULATION TO BE USED

A. Safety Data

1. Acute Mammalian Studies

a. Oral

Palmer and Radeleff studied the toxicity of atrazine and several other herbicides to cattle, sheep, and chickens. For atrazine tests, "cattle and sheep were dosed by either drench or capsule, chickens by capsule. The toxic dosage for cattle was 25 mg./kg. after 8 doses by drench and 2 by capsule. The toxic dosage for sheep was 5 mg./kg. No lesser dosage was tried. However, one sheep received 199 consecutive doses at 50 mg./kg. before it was poisoned and died. Chickens given 10 at 50 mg./kg. had a significant reduction in weight gains."

"To relate the toxic dosages found for cattle, sheep, and chickens to the application rates recommended for each herbicide," the authors "calculated the probable amounts that could be consumed daily from recently sprayed fields or pastures. In these calculations, we considered neither the influence of environmental factors such as soil type, temperature, and rainfall, nor the decomposition rates of the herbicides being studied.

"'The U.S.D.A. Summary of Registered Agricultural Pesticide Chemical Uses' was utilized for the application rates[1/]. An

1/ U. S. Department of Agriculture. 1966. U. S. D. A. Summary of Registered Agricultural Pesticide Chemical Uses. Ed. 2, Sup. III, 836 pp. (See also subsequent preliminary notices of U. S. D. A. pesticide summary entry to Dec. 15, 1967.)

arbitrary, although realistic, yield of 0.1 pound of air-dry forage per square foot of area was selected, which is equivalent to approximately 2 tons per acre. This would represent a high-quality, improved pasture. The reader must, of course, make adjustments for local conditions. A sparse cover of vegetation would allow more of the herbicide to reach the ground and be unavailable to animals, whereas a more lush vegetative cover would tend to hold more of the material available. In the latter case, however, less of the total forage of the area would be consumed in any one day.

"Further assumptions were: (1) that an animal would consume, as forage, 3 percent of its body weight each day; and (2) that all the chemical formulation applied would adhere to the vegetation. Although this latter is never actually the case, this assumption gives the maximum exposure to be expected.

"An application of 1 pound of chemical to 1 acre of land provides 10.4 milligrams for each square foot. We may simplify the whole calculation to a single statement that 1 pound actual of herbicide per acre provides a 7-milligram per kilogram (mg./kg.) dosage to the animal under the conditions here assumed to exist. Each 2.2 pounds of animal weight equals 1 kilogram or 1,000 grams. In turn, 1 pound equals 454 grams. The equivalent of 1,000 mg./kg. is 454 milligram per pound (mg./lb.)."

"Application rates for atrazine range from 0.4 to 6.4 pounds actual per acre. Rates of less than 1 pound would be hazardous for sheep. Rates of 3 pounds actual per acre would be hazardous for cattle. The 6.4 pound rate would be hazardous for chickens." (Palmer, J.S. and R. D. Radeleff. 1969. The toxicity of some organic herbicides to cattle, sheep, and chickens. U. S. D. A. A. R. S. Production Research Report No. 106. 26 p.)

5. Stability

a. Atrazine is a relatively stable compound, but is subject to decomposition by ultraviolet irradiation. However, under normal field conditions this effect would be small.

b. Microbial action probably accounts for the major breakdown of atrazine in the soil. A range of soil micro-organisms can utilize it as a source of energy and nitrogen. The effects of atrazine on these and other organisms appear to be small.

References

1. Bryant, J. B. 1963. Bacterial decomposition of some aromatic and aliphatic herbicides. Ph.D. Thesis Pennsylvania State University, University Park.
2. Volk, G. M. and C. F. Eno. 1962. The effect of simazine and atrazine on certain of the soil microflora and their metabolic processes. Florida Soils and Crop Sci. Soc. 22: 49-56.

c. 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 direct importance in atrazine dissipation under most field conditions. Atrazine is more subject to UV and volatility losses than simazine, but probably about equal or less subject to these losses compared to the commercial methylmercapto- or methoxytriazines.

References

1. Foy, C. L. 1964. Volatility and tracer studies with alky-lamino-s-triazines. Weeds 12: 103-108.
2. Jordan, L. S., B. E. Day and W. A. Clerex. 1964. Photodecomposition of triazines. Weeds 12: 5-6.
3. Kearney, P. S., J. T. Sheets, and J. W. Smith. 1964. Volatility of seven s-triazines. Weeds 12: 83-87.

d. Atrazine is very stable over several years of shelf life, with only slight sensitivity to natural light and extreme temperatures which would occur normally.

B. Phytotoxicity, Translocation, and Persistence In Plants

1. Atrazine is absorbed through both roots and foliage, although foliar absorbtion is often small in most plants under field conditions, depending on species, environmental conditions, et cetera.

The herbicide can be washed off plant foliage by rain. Following absorption through roots and foliage, it is translocated acropetally in the xylem and accumulates in the apical meristems. It acts as a photosynthetic inhibitor, but may have additional effects.

Atrazine is readily metabolized by tolerant plants to hydroxy-atrazine, which in turn is further degraded to CO_2 and other metabolities. This non-enzymatic alteration of atrazine is a major protective mechanism in most crops where it is used. Soil placement selectivity is also important in the case of some deep rooted perennial crops. Atrazine accumulates in sensitive plants, causing chlorosis and death.

Limited studies have shown some minor fungicidal and nematocidal activity but no insecticidal activity.

References

1. Funderburk, H. H. and D. E. Davis, 1963. The metabolism of C 14 chain-and ring-labeled simazine by corn and the effect of atrazine on plant respiratory systems. Weeds 11: 101-104.
2. Gysin, H. and E. Knusli. 1960. Chemistry and herbicidal properties of triazine derivatives. Advance. Pest. Control Res. 3: 289-358.
3. Hilton, J. L., L. L. Jansen, and H. M. Hull. 1963. Mechanisms of herbicidal action. Ann. Rev. Plant Physiol. 14: 353-384.
4. Montgomery, M. and V. H. Freed. 1964. Metabolism of triazine herbicides by plants. J. Agr. Food Chem. 12: 11-14.

C. Persistance In Soils

1. Adsorption and Leaching Characteristics In Basic Soil Types

a. 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 on temperature, moisture, pH, etc. Atrazine is not normally found below the upper foot of soil in detectable quantities, even after years of continuous use.

The residual activity of atrazine in soil at selective rates for specific soil types is such that most rotational crops can be planted one year after applications, except under an arid or semiarid climate. Atrazine will persist longer under dry and cold conditions or conditions not conducive to maximum chemical or biological activity. Broadcast rates needed in some of the heavier organic matter soils of the North Central states result in enough residue carry over, under some conditions, to injure small grains, alfalfa, and soybeans planted 12 months later. Plant removal and chemical alteration are also factors in dissipation.

References

1. Ashton, F. M. 1961. Movement of herbicides in soils with simulated furrow irrigation. Weeds 9: 612-619
2. Talbert, R. E. and O. H. Fletchall. 1964. Inactivation of simazine and atrazine in the field. Weeds 13: 33-38
3. _____1965. The adsorption of some s-triazines in soils. Weeds 13: 46-52
4. Gysin, H. and E. Knusli. 1960. Chemistry and herbicidal properties of triazine derivatives. Advance. Pest. Control Res. 3: 289-358.

REPORT

ON

BACKGROUND INFORMATION

FOR

CACODYLIC ACID

I. GENERAL INFORMATION

A. Common Name. Cacodylic acid.

B. Chemical Name. Dimethylarsinic acid.

C. Registered Uses.

1. For post-emergent weed control.

2. For conifer and hardwood control.

3. For bark beetle suppression and prevention. This use is registered only for application by professional foresters in forestry management programs in the Rocky Mountains of South Dakota, Colorado, Arizona, and New Mexico.

D. Formulation Manufactured. Silvisar 510 Tree Killer--a solution containing 6.0 lbs. of dimethylarsinic acid equivalent per gallon. (See Table 1 for materials used for post-emergent weed control.)

E. Dilution of Formulation for Use. Silvisar 510 is entirely soluble in water and can be mixed with water to form diluted solutions. Half-strength Silvisar 510--full strength Silvisar 510 mixed with an equal amount of water--has proven effective in bark beetle suppression and prevention.

F. Rates and Methods of Application.

1. Conifer and Hardwood Control (Silvicide). Full-strength Silvisar 510 is injected into undesirable trees by two methods:

a. Ansul "Hypo-Hatchet" Injection. This hatchet-like unit cuts and injects in one operation. The injector works by inertia and is calibrated to inject at least one milliliter of chemical per stroke. Rates for this method are:

(1) Conifers and Hardwood - Growing Season. For trees below 8 inches diameter at breast height (d.b.h.), make one cut per 2 inches of d.b.h. (4½" spacing between cut edges) at waist height or below. For trees 8 inches d.b.h. and larger, make one cut per 1 inch d.b.h. (1½" spacing between cut edges).

(2) Conifers - Dormant Season. Make one cut per 1 inch of d.b.h. (1½" spacing between cut edges) at waist height or below.

Table 1.--Solutions containing cacodylic acid that are registered for uses other than for a silvicide or systemic insecticide.

Trade name	Manufactured by	Lbs. cacodylic acid per gal.	USDA or EPA Reg. No.	Use
RAD-E-CATE 35	Vineland Chemical Co.	3.25	2853-28	Post-emergent, non-selective grass and weed killer
CHEX-MATE	Vineland Chemical Co.	1.25 + 3.00 lbs. MSMA	02853 50001 AA Calif. Reg. No. only	Post-emergent, non-selective general weed control
Phytar 560	The Ansul Co.	2.48	6308-20	General post-emergent weed control
Phytar 138	The Ansul Co.	65% CA (Powder formulation)	6308-11	General post-emergent weed control
Broadside	The Ansul Co.	1.25 + 3.00 lbs. MSMA	6308-65	General post-emergent weed control

(3) Hardwoods - Dormant Season. Make a complete frill at waist height or below.

b. Spaced-Cut Application. A hatchet or similar cutting tool can be used to make the cut and Silvisar 510 added to the cut with a plastic squeeze bottle or pump-type oil can other than those made of zinc, tin, or aluminum. Rates for this method are:

(1) Conifers and Hardwoods - Growing Season. For trees below 8 inches d.b.h., apply 1 milliliter of Silvisar 510 per cut per 2 inches of d.b.h. (6" spacing between cut centerlines) at waist height or below. For trees 8 inches d.b.h. and larger, use 1 to 2 milliliters per cut per 1 inch d.b.h. (3" spacing between cut centerlines).

(2) Conifers - Dormant Season. Apply 1 milliliter of Silvisar 510 per cut per 1 inch of d.b.h. (3" spacing between cut centerlines).

(3) Hardwoods - Dormant Season. Apply 1 milliliter of Silvisar 510 per cut in a complete frill at waist height or below.

2. Bark Beetle Prevention and Suppression. A complete, trough-like frill is made around the entire tree within 18 inches of the ground using a hand hatchet or small ax. A plastic squeeze bottle is used to apply 1 milliliter of chemical evenly in the frill for each inch of tree circumference.

a. Pre-Flight Treatment - Dendroctonus rufipennis Only.

(1) Fall Treatment. Trees are frilled and treated with half-strength or full-strength Silvisar 510 in October and felled 4 weeks after treating.

(2) Spring Treatment. Trees are frilled and treated with half-strength Silvisar 510 4-8 weeks before peak beetle emergence and felled 2-4 weeks after treating.

b. Pre-Harvest Preventive Treatment - D. rufipennis, D. ponderosae, D. pseudotsugae, D. adjunctus, Ips lecontei, I. pini, and I. confusus. Trees are frilled and treated with full-strength Silvisar 510 at least 4 weeks before cutting. A minimum of 4 weeks should be allowed between treating and felling.

c. Post-Flight Treatment - D. rufipennis, D. ponderosa, D. pseudotsugae, D. adjunctus, I. lecontei, I. pini, and I. confusus Trees are frilled and treated with full-strength Silvisar 510 within 2-3 weeks after beetle attack.

G. Tolerances in Food or Feed and Other Safety Limitations. The following tolerances have been granted for cacodylic acid expressed as As_2O_3:

2.8 ppm in cotton seed
1.4 ppm in kidneys and livers of cattle
0.7 ppm in meat, fats, meat by-products except kidney and liver

Silvisar 510 forms arsine gas when it comes in contact with zinc, tin, or aluminum; therefore, this material should not be stored or applied in containers made of these metals. Silvisar 510 is moderately corrosive; therefore, injection equipment should be thoroughly rinsed with water immediately after use.

H. Manufacturer or Producer

The Ansul Company
Marinette, Wisconsin 54143

II. TOXICITY DATA ON FORMULATION TO BE USED

A. Safety Data[1/]

1. Acute Mammalian Studies

a. Oral

(1) Estimated Acute LD50

(a) Technical Grade Cacodylic Acid. 77% cacodylic acid (CA); 0.7 g per kg (adult male albino rats); WARF Institute (Wisconsin Alumni Research Foundation Laboratories, Madison, Wisconsin).

(b) Technical Grade Sodium Cacodylate (NaCA). 30% CA equivalent; 4.3 g (NaCA) per kg (adult male albino rats): WARF Institute.

(c) Ansar 160. Sodium cacodylate 24.78%; CA equivalent 30.13%; sodium chloride 8.70%; 3.2 cc per kg (adult albino rats); WARF Institute.

(d) Phytar 560. 23.4% CA equivalent; 254.0 mg/kg (Holstein dairy calves); E. S. Erwin & Associates, Phoenix, Arizona.

1/ Except where otherwise noted, toxicity data was summarized from a report by The Ansul Company, Marinette, Wisconsin, entitled "Toxicological Data - Methanearsonic Acid and Dimethylarsinic Acid," June 5, 1967, with an addendum dated October 10, 1969.

(e) Ansar 160. 24.78% sodium cacodylate; 8.76 CA; 30.13% CA equivalent; 200.0 mg/kg (Holstein dairy calves); E. S. Erwin & Associates, Phoenix, Arizona.

(2) Acute Oral LD_{50}

(a) Technical Grade Cacodylic Acid. 61.3% CA; 1.40 g per kg (young male albino rats); 1.28 g per kg (young female albino rats); Industrial Bio-Test Laboratories, Northbrook, Illinois.

(b) Phytar 560. 23.4% CA equivalent; 2.6 g (560) per kg (young male and female albino rats); Industrial Bio-Test Laboratories.

(c) Silvisar 510. 50.0% CA; 1.8 g per kg (young male albino rats); 1.0 g per kg (young female albino rats); Industrial Bio-Test Laboratories.

(3) Acute Oral LD_{100}

(a) Technical Grade Sodium Cacodylate. 1.23 g per kg (dairy calves); E. S. Erwin & Associates.

(b) Phytar 560. 2.0 g per kg (dairy calves); E. S. Erwin & Associates.

b. Eye and Skin Irritation

(1) Dermal Irritation

(a) Ansar 160. 24.78% sodium cacodylate; 8.76% CA; 30.13% equivalent; non-irritating to the skin in 72-hour exposure (albino rabbits); WARF Institute.

(b) Technical Grade Cacodylic Acid. Non-irritating to the skin in 24-hour exposure (albino rabbits); WARF Institute.

(2) Eye Irritation - Technical Grade Cacodylic Acid. Non-irritating to the eyes in 2-4 sec. exposure (adult albino rabbits); WARF Institute.

2. Subacute Studies

a. Oral

(1) Technical Grade Cacodylic Acid. Technical grade cacodylic acid was fed to weanling male rats at 700, 1400, and 2800

ppm in the basal ration daily for 3 weeks. Substantial drop in food consumption and weight gain at 2800 ppm. Evidence of reduced activity of spermatogonia cells with some atrophic changes of the seminiferous cells at 2800 ppm. One animal showed some early degeneration of the hepatic cells. No such findings at 700 and 1400 ppm. WARF Institute.

(2) Phytar 560. 560 was fed at 200, 400, and 1200 mg per kg in 8 pounds of supplemental cottonseed meal to each of two Holstein calves at each level, each day for 1 week. 200 mg per kg - calves quit feeding on 7th day; 400 mg per kg - calves quit feeding on 6th day; 1200 mg per kg - one calf quit feeding on 5th day; 1200 mg per kg - one calf died on 3rd day. Only one calf of six showed diarrhea. Remaining five calves recovered completely on normal ration for 7 days. E. S. Erwin & Associates.

(3) Ansar 138. A 60-day feeding test on the metabolism of cacodylic acid (Ansar 138) in dairy cows was conducted. Two Holstein milk cows were fed a diet of ground barley, wheat bran, and cottonseed meal containing 10 ppm of cacodylic acid. This resulted in a daily intake of 24.5 mg/kg/cow. In another group of cows, an equal weight of arsenic acid was fed to cows. The milk from both groups of these cows was analyzed and found to contain no arsenic during the entire test period. The excretion of arsenic is primarily by way of the urine, and a balance between intake and output is present after 30 days of feeding. At the end of the experiment, the cows were sacrificed and 10 tissues and bone were analyzed for arsenic. It was concluded that no tissues stored arsenic compounds on a cumulative basis, even though fractional parts per million of arsenic were detected in the liver, spleen, and pancreas. The differences in arsenic content of the organs from the cows fed cacodylic acid and those fed arsenic acid were insignificant (Peoples 1964).

(4) Pure Cacodylic Acid. Cacodylic acid was fed at 3, 15, and 30 ppm to dogs, and at 3, 15, and 100 ppm to rats, in the basal ration for 90 days. No-effect level for dogs - 30 ppm. No-effect level for rats - 100 ppm. WARF Institute.

b. Dermal - Technical Grade Cacodylic Acid. Technical grade cacodylic acid, in the form of an aqueous suspension, was applied by rubbing to the clipped (area of about 4 x 6 inches) trunks of adult male albino rabbits, at levels of 1.0, 1.6, 2.5, 3.9, 6.0, and 9.4 g per kilogram. Two rabbits were used at each level, with the skin abraded on one animal and intact on the other. A rubber sleeve was placed over the treated area. Exposure was for 12 hours overnight, after which the sleeve was removed, the animal wiped clean and returned to its cage. Treatment was for 5 days per week for 3 weeks, followed

by a 2-week post-treatment observation period. Dermal LD_{100} - 1.0 g per kg for abraded animals; Dermal LD_{100} - 2.5 g per kg for intact animals; Dermal LD_0 - 1.6 g per kg for intact animals. WARF Institute.

3. Other Studies

a. Carcinogenicity. Arsenic has only been associated with poisoning and was indicated quite early as a carcinogen. More evaluations suggest that the early tests reporting arsenic-induced carcinoma were inadequate. Frost (1970) cites numerous studies which attempted but failed to demonstrate arsenic-induced carcinoma. Cacodylic acid was placed in group c4 by the Secretary's Commission on Pesticides and their relationship to environmental health (Mrak 1969). This group contains pesticides which were judged not positive for carinogenicity in one species (mouse), but current guidelines require negativity in two species. The commission gave this group a moderate priority for testing, but felt no changes in practices in the field were warranted.

S. S. Pinto and B. M. Bennett (1963) believe that it is a mistake to make blanket condemnations of the use of arsenic without first looking at the data. He has reviewed the early literature on human tumors from arsenic and also the recent opinions and interpretations of these early papers. There is reason to believe that the "arsenic tumors" observed in 1820 may have been due to other causes such as selenium poisoning. He reviewed the medical histories and causes of death of the long-term employees of a copper smelting company producing arsenic trioxide. He showed that the workers do excrete high levels of arsenic, but that their incidence of cancer is no greater than for other persons in the State of Washington. He concluded that there is no evidence that exposure of these workers to arsenic trioxide is a cause of systemic cancer in humans. In a sense, this amounts to the use of human guinea pigs for establishing the lack of carcinogenicity of arsenic trioxide.

b. Mutagenicity. Cacodylic acid is a mitotic poison in mammalian organisms. King and Ludford (1960) found that injections in mice produced "profound disturbances of cell division" and it "stimulated mitosis in cells of the crypts of Lieberkuehn" and of transplanted tumors. The significance of this finding in terms of exposure to cacodylic acid in the field is not known.

c. Teratogenicity. Cacodylic acid is considered to be a teratogenic agent, producing abnormalities during embryonic development. There are several references to this type of action, although only two examples are quoted. Salzgeber (1955) observed teratogenic effects in 10-day chick embryo genital organs cultured in vitro and has reported

that the greatest damage is to the cortical region. Rostand (1950) has treated tadpoles of Rana temporia with solutions of cacodylic acid for 3 weeks when the hind legs were in the process of development, and abnormalities were observed at 0.01% of sodium cacodylate. (This concentration is 100 ppm and is equivalent to 270 lb/acre ft of water.)

Additional testing, using the techniques reported by Mrak (1970), is needed. Relation of these reports of teratogenic potential and field use of the chemical require further investigation.

d. Fish Toxicity

(1) Sodium Cacodylate (NaCA). 30% CA equivalent; TLm (median tolerance limit) at 96 hours -- 750 ppm (bluegill sunfish). Louisiana Wildlife & Fisheries Commission.

(2) Phytar 560. 23.4% CA equivalent; TLm at 96 hours -- 80 ppm (bluegill sunfish). Louisiana Wildlife & Fisheries Commission.

(3) Other Studies. K. H. Oliver (1966) exposed Gambusia addinis (mosquito fish), Notropis maculatus (tail-light shiner), and Micropterus salmoides (largemouth black bass) to concentrations ranging from 100 to 10,000 ppm of cacodylic acid for periods up to 72 hours. All three species of fish survived the 100 ppm level for this period. Although there were some deaths of the Gambusia at lower doses, 12 out of 20 survived 631 ppm for 72 hours. Five out of 10 of the Notropis survived exposure to 631 ppm for 72 hours. In a similar experiment with tadpoles, it was shown tadpoles (Bufo terrestris) survived the 100 ppm level for 48 hours, and all died at this time period at 1,000 ppm. The Bureau of Commercial Fisheries (1966) showed that cacodylic acid at 40 ppm has no effect in 48 hours on pink shrimp (Penaeus duorarum), eastern oyster (Crassostrea virginica), or longnose killifish (Fundulus similis).

e. Game Bird Toxicity. Mallard ducks and chukar partridge were dosed at levels of Silvisar 510 (50% CA) up to 2000 mg/kg. All birds survived, but some showed symptoms of intoxication at higher dosages. U.S. Department of Interior, Fish and Wildlife Service.2/

f. Chicken Feeding Studies. Pure cacodylic acid was fed at 0.6, 6.0, and 60 ppm in the basal ration of 7 female and 3 male leghorn chickens at each level, for 10 weeks. No significant arsenic residues in eggs, lean meat, liver, and kidneys at 0.6 and 6.0 ppm. No residues in fat at all levels. Some small but definite arsenic

2/ Memo from Jack F. Welch, Director, Bureau of Sports Fisheries and Wildlife, Fish and Wildlife Service, U.S. Dept. of Interior, Denver, Colorado, to F. Leroy Bond, Assistant Regional Forester, USDA Forest Service, Albuquerque, New Mexico, dated March 20, 1970.

residues in eggs, lean meat, liver, and kidneys at 60 ppm. One week post-feeding on basal ration only removed residues in liver and kidneys, and reduced level in lean meat, for 60 ppm level. WARF Institute.

B. Physical-Chemical Properties[3/]

1. Boiling point - +110° C.

2. Flash point - none

3. Physical state - crystalline

4. Density - 1.44 gram/ml.

5. Vapor pressure - same as water

6. Solubility - 200 g/100 g water; 26 g/100 g alcohol. Insoluble in ether.

7. Stability somewhat hygroscopic. Stable to fuming nitric acid, aqua regia, hot $KMnO_4$ sol.

8. Melting point - 195-196° C.

III. EFFICACY DATA UNDER FIELD AND LABORATORY CONDITIONS

A. Effectiveness for Intended Purpose When Used as Directed

1. As a Systemic Insecticide for Bark Beetle Suppression and Prevention.

a. Post-Flight Treatment. Chansler and Pierce (1966) pioneered the investigations into the use of cacodylic acid treatment for bark beetle suppression. They injected undiluted Ansar 160 herbicide (a solution manufactured by The Ansul Company containing the equivalent of 3.25 lbs. of cacodylic acid per gallon) and Silvisar 510 Tree Killer directly into the sap streams of individual ponderosa pine infested with Dendroctonus adjunctus and D. ponderosae, Douglas-fir infested with D. pseudotsugae, and Engelmann spruce infested with D. rufipennis, soon after attack. Population reductions from this treatment ranged from 84-99 percent. Chansler et al. (1970) treated

3/ Data obtained from TSI Company, Flanders, New Jersey.

ponderosa pine with undiluted Silvisar 510 within 2 weeks after they had been infested by D. ponderosae and obtained almost complete beetle reduction. D. rufipennis broods were significantly reduced when newly infested trees were treated with undiluted Silvisar 510 (Buffam 1969a). Buffam and Flake (1971) obtained 100 percent mortality of D. adjunctus broods when recently infested ponderosa pines were treated with undiluted Silvisar 510. Ollieu (1969) obtained 97 percent reduction of D. frontalis brood when pines were treated with Silvisar 510 at 1-2 days after attack. Brood reduction was only 59 percent when trees were treated 3-4 days after attack.

b. Pre-Attack Treatment. Several studies have been made to determine the effectiveness of cacodylic acid-treated trees as lethal traps. I. lecontei was attracted to ponderosa pine treated with Silvisar 510 and felled 4 weeks later (Buffam 1969b). However, significantly more attacks were found in non-treated, felled trees. Beetle mortality in treated trees averaged 94 percent, while that in non-treated was less than 1 percent. Stelzer (1970) found that density of attack and subsequent mortality of brood and attacking adults of I. lecontei varied considerably with the time of year the trees were treated. D. ponderosae attracted to ponderosa pine treated with undiluted Silvisar 510 prior to the attack period were unable to produce brood (Chansler et al. 1970). Very few D. rufipennis brood were produced in Engelmann spruce trees treated with undiluted Silvisar 510 at least 4 weeks before felling (Buffam and Yasinski 1971). Frye and Wygant (1971) treated Engelmann spruce with undiluted Silvisar 510 and felled the trees 9-14 days later. D. rufipennis brood development was prevented in the treated trees. Buffam (1971) tested different treatment times and dosage rates to determine the most effective combination against D. rufipennis. Half-strength Silvisar 510 was as effective as full-strength in reducing brood development. Engelmann spruce treated in mid-June and felled 2 weeks later were as effective in attracting D. rufipennis as non-treated trees. Few survivors were found in treated trees, whereas significant numbers were found in non-treated trees.

Williamson (1970) obtained decreased brood survival in pines treated with cacodylic acid before attack by the southern pine beetle. Williamson (1971) suggests a pest management system for the southern pine beetle in which the synthetic attractant Frontalure is used to attract beetles to cacodylic acid-treated trees. This method has also been suggested for suppression of the spruce beetle (Anonymous 1971b). The Frontalure-cacodylic acid treatment was tested in loblolly pine stands in Virginia in 1971 for control of the southern pine beetle (Morris and Capony 1971). This method resulted in a 62 percent reduction in beetle populations.

McGhehey and Nagel (1967) checked western hemlock trees killed with a 90 percent solution of Silvisar 510 during thinning operations. They found that neither Pseudohylesinus grandis or P. tsugae survived in treated trees. Oliver (1970) found that D. brevicomis broods survived in injected trees, and attacked and killed six leave trees. Newton and Holt (1971) found that brood of D. ponderosae and I. pini were not able to survive applications of cacodylic acid, monosodium methanearsonate (MSMA), and a mixture of cacodylic acid and MSMA during precommercial thinning operations in ponderosa pine.

c. General. Little is known of the mode of action of cacodylic acid in killing bark beetles. Chansler and Pierce (1966) postulated that treatment may kill the cambium layer and make the habitat unfavorable for the insect, or this material may have direct insecticidal properties. A study reported by the Southern Forest Research Institute (Anonymous 1971a) showed that cacodylic acid was not toxic to adult southern pine beetles when applied topically in concentrated form. Newton and Holt (1971) report that reduction of organic arsenicals to arsines is a possible explanation for mortality. Frye (1970) added Silvisar 510 to ground phloem and then placed this in test tubes along with D. rufipennis males and females. Beetle mortality ranged from 16 percent with the 0.06 percent solution to 100 percent at the 0.5, 4, and 10 percent solutions after 10 days of exposure. Frye and Wygant (1971) speculated that cacodylic acid treatment might break down the carbohydrate food source and alter phloem pH, thus making an unfavorable environment for the spruce beetle.

Bark beetles often carry blue stain fungus into attacked trees. The sapwood of infested trees normally becomes colonized by this stain within 1 or 2 years. Frye and Wygant (1971) found that blue stain was very light in treated trees and heavy in untreated trees. Hinds and Buffam (1971) found that stain had penetrated the sapwood of untreated trees, but was negligible in treated trees 1 year after treatment.

Associated insects are often not affected by cacodylic acid treatment. Frye and Wygant (1971) noticed that egg gallery construction by the ambrosia beetle, Trypodendron lineatum, was not impaired in treated Engelmann spruce. Hinds and Buffam (1971) also found ambrosia beetle galleries to be common in treated Engelmann spruce. Flatheaded borer larvae were found in ponderosa pine treated with MSMA, cacodylic acid, and a mixture of both (Newton and Holt 1971). McGhehey and Nagel (1967) felt that the cacodylic acid treatment in hemlock had no adverse effect on parasites and predators because larvae of the fly, Medetera aldrichii, were found in larval mines and adults of the wasp, Cecidostiba acuta, emerged from treated trees.

2. As a Silvicide

Experiments with cacodylic acid for thinning began in 1963 in New Zealand (Harrison-Smith 1963). Cacodylic acid was added to holes made by a boring machine to thin stands of Monterey pine. Hedderwick (1966) treated stems of Pinus radiata and P. patula in New Zealand with cacodylic acid. He concluded that cacodylic acid was a comparatively safe and effective substitute for sodium arsenite when used in solution at high concentrations. At low concentrations, cacodylic acid was little more efficient than ammonium sulphamate and was nine times more expensive. Day (1965) injected cacodylic acid into red maple, aspen, paper birch, sugar maple, ironwood, serviceberry, and jack pine, and concluded that this material has considerable potential as a silvicide. Smith (1966) reported results of studies in 1964 and 1965 where cacodylic acid was tested against jack pine and red pine. Almost complete crown-kill was obtained with this material. Smith (1966) also reported results of a study by Welton and Theiler, Bureau of Indian Affairs, Lame Deer, Montana. In this study, almost complete crown-kill of ponderosa pine was obtained with injection of a 30 percent solution of cacodylic acid. Smith (1965) said that 90-100 percent crown-kills and defoliation of red maple, hickories, aspen, paper birch, hawthorn, pin cherries, American beech, red oak, and other hardwoods occurred from frill injections of Ansar 160. Bore-hole injections of Ansar 160 into quaking aspen, red maple, paper birch, and red oak resulted in excellent results, except with red oak. Smith (1965) concluded that American elm, American basswood, and Eastern hop hornbean can be crown-killed by a oneshot injection of Ansar 160 during the growing season as a result of a test by the Kimberly-Clark Corporation.

Injection of a 25 percent aqueous solution of cacodylic acid gave 100 percent kill of Douglas-fir, cherry, willow, and hawthorne, and relatively poor control of bigleaf maple in studies by Newton (1964). Injector treatments of Tordon, 2,4-D, cacodylic acid, and a mixture of 2,4-D, 2,4,5-T, and TBA were tested by Newton (1965). Tordon gave the best kill of Douglas-fir, followed by cacodylic acid. Cacodylic acid tended to concentrate in terminal whorls of branches, thus killing tops but not the entire trees in many cases. Newton and Holt (1967b) injected cacodylic acid into ponderosa pine at four different seasons and at three dosage rates. The response to treatments in September and December was much less than that to treatments in March and June. "Virtually any treatment during spring months appeared to produce good results." Newton and Holt (1967c) injected undiluted endothall, an endothall and Silvex mixture, and cacodylic acid into Oregon oak, bigleaf maple, and red alder. "None of these materials were completely effective on all species, although cacodylic acid appeared to be the best general defoliant." Newton and Holt (1967a) also tested cacodylic acid against lodgepole pine at different seasons and with three dosage

rates. "Generally, lodgepole pine appears to be very sensitive to cacodylic acid. Limits of effectiveness appear to be imposed by lateral translocation restrictions, since all tissue within the apparent range of herbicide movement was badly damaged, regardless of dosage." Newton (1967) injected Douglas-fir trees with several herbicides at several dosage rates--cut spacings--and found that picloram, Tordon, and cacodylic acid gave the best results. Newton and Webb (1970) state that cacodylic acid and MSMA are effective in killing young ponderosa pines any season of the year. They also state that of the two herbicides, MSMA is cheaper and more effective.

Newton (1968) summarized the work with cacodylic acid. Injections of this material gave excellent results against bitter cherry; good results against alder, Douglas-fir, grand fir, lodgepole pine, Oregon white oak, and ponderosa pine; fair to good results against western hemlock. When mixed with MSMA, the results were excellent against Douglas-fir, and good against lodgepole pine and ponderosa pine. Top-kill of Sitka spruce was obtained with cacodylic acid. Oliver (1970) reported that injections of cacodylic acid into ponderosa pine, Douglas-fir, red fir, and white fir resulted in inadequate thinning in two of the three test stands.

B. Persistence in Soil, Water, or Plants. See Section IVB.

C. Compatibility with Other Chemicals. Cacodylic acid is compatible with MSMA.

IV. ENVIRONMENTAL IMPACT

A. Effects on Non-Target Organisms. Sollman (1950) describes cacodylic acid as a material which has medicinal properties similar to those of inorganic arsenic "to which it is partly reduced in the body." Since the reduction is slow the toxicity is reduced in the body." Preliminary experiments by Peoples (1964) are contradictory and indicate that no reduction to trivalent arsenic occurs since administration of cacodylic acid to cows, followed by analysis of tissues, showed only the pentavalent arsenic to be present. Cacodylic acid, especially when given by mouth, imparts a garlic odor to the breath, sweat, and urine. The dosages which have been given to humans as pills or as hypodermic injections vary from 0.025 to 0.15 g/day; Sollman (1950) adds, however, that cacodylate is not effective in the chemotherapy of syphilis, bacterial, or parasitic infections.

Peoples (1964), working with pentavalent inorganic arsenic acid, found 76-98 percent of daily dose excreted in urine by cows during a 7-week feeding study. Similarly, little to no arsenic was recovered from various animal tissues.

Tarrant and Allard (1972) (see Norris 1971) studied the excretion of arsenic in urine from forest workers using cacodylic acid as a silvicide. Significant quantities of arsenic in urine from certain individuals suggests uptake of this chemical through the skin occurs and dermal exposure should be avoided.

The toxicity of cacodylic acid in humans is not known, but workers in The Ansul Chemical Company plant have had repeated exposures over long periods of time. The Ansul Company says that their experience confirms the observations on rats that the toxicity of these compounds is "relatively low" (Stevens 1966). Norris (1971) concluded the safe use of organic arsenicals depends on minimizing exposure of applicators and animals in treated areas.

Morton et al. (1972) fed herbicides to the honeybee, Apis mellifera, in 60 percent sucrose syrup at concentrations of 0, 10, 100, and 1000 parts per million by weight. Cacodylic acid was extremely toxic at 100 and 1000 ppmw and moderately toxic at 10 ppmw.

B. Residues in or on Food or Feed or Entering into Food Chain via Air, Water, Soil, Plants, or Animals. Cacodylic acid, methanearsonic acid, and their salts are contact-type, post-emergence herbicides. Ehman (1965) has reported that when pasture lands are treated with 5 lbs. of cacodylic acid, and planted to alfalfa and rye grass within 3 days after treatment, growth was not inhibited and cuttings from these two crops did not show arsenic residues.

Ehman (1965) found that when a combination of 10 lb/acre of cacodylic acid and 10 lbs. of DSMA were used in grapefruit orchards, no residues could be found in the fruit. In soil build-up tests, utilizing 15, 22, 41, and 79 lb/acre of DSMA, no arsenic residues were found in grapefruit.

Ehman and Birdsall (1963) reported on a study that involved the residual effects of cacodylic acid on beans, potatoes, carrots, cabbage, corn, and soybeans. The test plots were sprayed with 1 gal. of a cacodylic acid solution. The treatment was equivalent to 5 lb/acre of pure cacodylic acid. The plots were planted 5 days later. Over a period of 1 month, 8.9 inches of rainfall were applied. The increase in soil arsenic by analysis was 3 ppm at a 3-inch depth. The authors stated, "There was no significant pickup of arsenic by edible crops in the treated plots." Unfortunately, no data for control plots were presented.

Newton (see Norris 1971) treated conifers with organic arsenicals in a thinning study in November. Foliage samples contained 110 ppm, 139 ppm, and 58 ppm the following April, June, and August, respectively. Allard (see Norris 1971) measured

116 ppm As in dead pine needles and 2.5 ppm As in green needles from a treated tree. These data indicate needle fall from treated trees is a significant source of arsenic which will enter the forest floor. Norris (personal communication) finds MSMA and cacodylic acid are leached fairly quickly through 3-inch columns of chopped ponderosa pine, Douglas-fir, or mixed true fir-larch needles. In soil, he finds MSMA is quite resistant to leaching. Cacodylic acid is more mobile, but not to the extent that contamination of ground water is a problem.

Newton (1971) has reviewed the metabolism of the organic arsenicals and suggests that arsine or alkyl arsine are logical products of the microbial metabolism of MSMA and cacodylic acid. While the arsines are fairly toxic, they are also gases and would be expected to leave treatment areas in low concentrations in mass air movement. The production of arsine analogs under field conditions has not been demonstrated.

A number of studies have examined the soil behavior of MSMA and cacodylic acid. Dickens and Hiltbold (1967) showed DSMA was extensively adsorbed by various soils from water solutions of the herbicide. Soils with higher clay content adsorbed more DSMA. No DSMA leached through a 10-inch column of clay soil with 20 inches of water, while 52 percent of applied DSMA leached through a 10-inch column of loam. The remainder of the herbicide appeared to be tightly bound to the soil. Dickens and Hiltbold (1967) also demonstrated up to 16 percent dimethylation of DSMA in soil in 30 days. Woolson et al. (1969) reports organic and inorganic arsenic behavior similarly in soil. They find soils high in aluminum and iron bind arsenic tightly and reduce its availability, in a sense, detoxifying the arsenic. They show for instance the water soluble (available) arsenic level in a clay loam is decreased by 90+ percent in 4 weeks after application.

Ehman and Birdsall (1963) studied the adsorption of cacodylic acid on pasture sod. They sprayed the sod (4 ft. x 4 ft. x 10 in.) with 3.81 g of Ansul's Ansar 138, containing 65 percent cacodylic acid, and 0.85 g of Emulphor 620 surfactant. The sods were watered with about $\frac{1}{2}$ inch of rainfall at 1, 2, and 4 weeks. Some of the sod clay samples leached arsenic in the first 24 hours. In general, the cacodylic acid was strongly bound to the clay, silt loam, and sand sods. After 1 week, the cacodylic acid became evenly distributed throughout a 10-inch depth.

Ehman (1965) found that when an amount of disodium methanearsonate (DSMA) equivalent to 28 lb/acre was applied to the top of a soil column which was leached with 60 inches of water, less than 10 percent of the applied DSMA showed up in the leachate. When sandy loam was used in

the soil column, the figure was less than 6 percent. In a similar experiment performed with 15 lb/acre of cacodylic acid, and using an extrapolation to 60 inches of leaching water, about 9 percent leached through the sand column and 6 percent for the sandy loam. It is evident that DSMA and cacodylic acid are largely inactivated by the soil.

LITERATURE CITED

Anonymous. 1971a. Means to suppress brood development of the southern pine beetle in trees baited with Frontalure. Progress Report - Southern Forest Res. Institute. Mar.-Apr. : 7.

Anonymous. 1971b. Spruce beetle attacks trees baited with frontalin. Progress Report - Southern Forest Res. Institute. Jul.-Aug. : 16.

Buffam, P. E. 1969a. Final Report - Pre- and post-flight treatments with cacodylic acid for control of the Engelmann spruce beetle on Mt. Taylor in 1967. Office Report, USDA, Forest Service, Southwestern Region. 4 p.

Buffam, P. E. 1969b. Results of an Ips lecontei-cacodylic acid study at Prescott, Arizona, in 1968. Office Report, USDA, Forest Service, Southwestern Region. 4 p.

Buffam, P. E. 1971. Spruce beetle suppression in trap trees treated with cacodylic acid. J. Econ. Entomol. 64(4): 958-60.

Buffam, P. E., and H. W. Flake, Jr. 1971. Roundheaded pine beetle mortality in cacodylic acid-treated trees. J. Econ. Entomol. 64(4): 969-70.

Buffam, P. E., and F. M. Yasinski. 1971. Spruce beetle hazard reduction with cacodylic acid. J. Econ. Entomol. 64(3): 751-2.

Bureau of Commercial Fisheries. 1966. Bioassay screening test on cacodylic acid. Gulf Breeze Lab., Fla.

Chansler, J. F., D. B. Cahill, and R. E. Stevens. 1970. Cacodylic acid field tested for control of mountain pine beetles in ponderosa pine. USDA Forest Service Res. Note RM-161. 3 p.

Chansler, J. F., and D. A. Pierce. 1966. Bark beetle mortality in trees injected with cacodylic acid (herbicide). J. Econ. Entomol. 59(6): 1357-9.

Day, M. W. 1965. Cacodylic acid as a silvicide. Michigan Quart. Bull. 47(3): 383-386.

Dickens, R., and A. E. Hilbold. 1967. Movement and persistence of methanearsonates in soil. Weeds 15: 299-304.

Ehman, P. J. 1965. The effect of arsenical buildup in the soil on subsequent growth and residue content of crops. Southern Weed Control Conf. Proc. 18.

Ehman, P. J., and J. J. Birdsall. 1963. Fate of cacodylic acid in soil and plants. Dept. of Army Contract DA-18-064-IML-2826(A). U.S. Army Biol. Lab., Fort-Detrick, Md.

Frost, D. V. 1970. Tolerances for arsenic and selenium: A psychodynamic problem. World Rev. of Pest Contr. Spring 197, 9(1): 6-27.

Frye, R. H. 1970. Spruce beetle mortality with cacodylic acid in Engelmann spruce trap trees. Master's thesis, Colorado State University, Fort Collins, Colo. 96 p.

Frye, R. H., and N. D. Wygant. 1971. Spruce beetle mortality in cacodylic acid-treated spruce trap trees. J. Econ. Entomol. 64(4): 911-16.

Harrison-Smith, J. L. 1963. Progress in poison thinning. New Zealand Timber J. Oct. 1963: 25-27.

Hedderwick, G. W. 1966. Cacodylic acid as an arborcide. New Zealand Pest Contr. Conf. Proc., 19th Conf: 165-169.

Hinds, T. E., and P. E. Buffam. 1971. Blue stain in Engelmann spruce trap trees treated with cacodylic acid. USDA Forest Service Res. Note RM-201. 4 p.

King, H., and R. J. Ludford. 1960. The relation between constitution of arsenicals and thin action on cell division. J. Chem. Soc. 2086-2088.

McGhehey, J. H., and W. P. Nagel. 1967. Bark beetle mortality in precommercial herbicide thinning of western hemlock. J. Econ. Entomol. 60(6): 1572-4.

Morris, C. L., and J. A. Copony. 1971. Frontalure-cacodylic acid tested for control of southern pine beetle on the eastern shore of Virginia. Progress Report - Southern Forest Res. Institute. Sep.-Oct. : 20.

Morton, H. L., J. O. Moffett, and R. H. MacDonald. 1972. Toxicity of herbicides to newly emerged honey bees. Environ. Entomol. 1(1): 102-104.

Mrak, Emil M. 1969. Report of the Secretary's Commission on pesticides and their relationship to environmental health. U.S. Dept. HEW.

Newton, M. 1964. Chemical control of conifers for pre-commercial thinning. Western Weed Contr. Conf. Res. Progr. Rep. 43-44.

Newton, M. 1965. Injector treatments for pre-commercial thinning of Douglas-fir. Western Weed Contr. Conf. Res. Progr. Rep. 42-43.

Newton, M. 1967. Influence of season and dosage on effectiveness of injections for control of Douglas-fir. Western Weed Contr. Conf. Res. Progr. Rep. 262.

Newton, M. 1968. Chemical silviculture. Symposium: Management of young growth Douglas-fir and western hemlock. Oregon State University, Corvallis, Ore. 21-29.

Newton, M. 1971. Organic arsenicals: Breakdown in forest trees and in media containing energy sources--a progress report to Environmental Protection Agency, Aug. 26, 1971. Oregon State University, Corvallis, Ore.

Newton, M., and H. A. Holt. 1967a. Response of lodgepole pine to injections of cacodylic acid. Western Weed Contr. Conf. Res. Progr. Rep. 268.

Newton, M., and H. A. Holt. 1967b. Response of ponderosa pine to injections of cacodylic acid. Western Weed Contr. Conf. Res. Progr. Rep. 267.

Newton, M., and H. A. Holt. 1967c. Tests of herbicides for multiple species control by injection. Western Weed Contr. Conf. Res. Progr. Rep. 270.

Newton, M., and H. A. Holt. 1971. Scolytid and buprestid mortality in ponderosa pines injected with organic arsenicals. J. Econ. Entomol. 64(4): 952-8.

Newton, M., and W. L. Webb. 1970. Herbicides and management of young pine. From symposium on regeneration of ponderosa pine held at Oregon State University, Corvallis, Ore., Sep. 11-12, 1969. 94-99.

Norris, L. A. 1971. Studies of the safety of organic arsenical herbicides as precommercial thinning agents: A progress report. Precommercial thinning in the Pacific Northwest. Washington State University, Pullman, Wash. p. 63-74.

Oliver, K. H. 1966. An ecological study on the effects of cacodylic acid on selected fauna and flora. Dept. of Army, Fort Detrick, Md. CDTL 45644.

Oliver, W. W. 1970. Cacodylic acid for precommercial thinning in mixed-conifer stands shows erratic results. USDA Forest Service Res. Note PSW-224. 3 p.

Ollieu, M. M. 1969. Evaluation of alternative southern pine beetle control techniques. Tex. Forest Serv. Publ. 104. 6 p.

Peoples, S. A. 1964. Arsenic toxicity in cattle. Ann. N.Y. Acad. Sci. III(2): 644-649.

Pinto, S. S., and B. M. Bennett. 1963. Effect of arsenic trioxide exposure on mortality. Arch. of Environ. Health 7: 583-591.

Rostand, J. 1950. Chemical teratogenesis in anurian batrachians. Compt. Rend. Soc. Biol. 144: 915-917.

Salzgeber, B. 1955. Modification observed in chick embryo genital organs inplanted in vitro after treatment with different teratogenic substances. Compt. Rend. Soc. Biol. 149: 190-192.

Smith, R. W. 1965. Preliminary report on "Ansar 160" (cacodylic acid) a potential one-shot silvicide. The Ansul Co., Marinette, Wisc. 11 p.

Smith, R. W. 1966. Progress report on cacodylic acid as a silvicide. Proc. Northeastern Weed Contr. Conf. 568-573.

Sollman, T. 1950. Manual of pharmacology and its application to therapeutics and toxicology. 7th ed. W. B. Saunders, Philadelphia 878-879.

Stelzer, M. J. 1970. Mortality of Ips lecontei attracted to ponderosa pine trees killed with cacodylic acid. J. Econ. Entomol. 63(3): 956-9.

Stevens, G. D. 1966. Weed control with dimethyl-arsinic acid. Proc. Southern Weed Contr. Conf. 19: 545-549.

Tarrant, R. F., and Jack Allard. 1972. Studies of the safety of organic arsenical herbicides as precommercial thinning agents: a progress report. Precommercial thinning in the Pacific Northwest. Washington State University, Pullman, Wash. p. 63-74.

Williamson, D. L. 1970. A pest management system for the southern pine beetle, Dendroctonus frontalis Zimmerman, in east Texas. Ph. D. Dissertation. Dept. of Entomol., Texas A&M University, College Station, Tex. 108 p.

Williamson, D. L. 1971. Management to reduce pine beetle infestations. Forest Farmer, Jan. 1971: 6, 7, and 18.

Woolson, E. A., D. C. Kearney, and J. H. Axley. 1969. Chemical distribution of arsenic in soil. Am. Chem. Soc. Mtg., N.Y., N.Y.

ENVIRONMENTAL STATEMENTS

BACKGROUND DOCUMENT

PESTICIDES

DICAMBA

F. W. Pond (R-1), Leader
R. Dalen (R-5)
H. Williston (SA)

JULY 1972

DICAMBA

I. General Information

A. Common name

Dicamba, Banvel, Banvel D

B. Chemical name

3,6-dichloro-o-anisic acid
(2-methoxy-3,6-dichlorobenzoic acid)

C. Registered uses

Postemergence weed control in field corn, wheat, oats, barley, sorghum, pasture/rangeland, perennial grass grown for seed, turfgrass, industrial brush control, and for noncropland areas such as fencerows, roadways, and wastelands.

D. Formulations manufactured

1. Banvel Herbicide - U.S.D.A. Reg. No. 876-25

Active ingredients	
Dimethylamine salt of 3,6-dichloro-o-anisic acid	49.0%
Dimethylamine salts of related acids	7.9%
Inert ingredients	
Water	43.1%

2. Banvel Brush Killer (oil soluble)

Active ingredients	
3,6-dichloro-o-anisic acid	44.5%
Related acids	6.6%
Inert ingredients	48.9%

3. Banvel 5G Granules - U.S.D.A. Reg. No. 876-103

Active ingredients	
3,6-dichloro-o-anisic acid	5.0%
Related acids	.9%
Inert ingredients	
Attapulgite clay	94.1%

4. Banvel 10G Granules

Active ingredients	
3,6-dichloro-o-anisic acid	10.0%
Related acids	1.8%
Inert ingredients	
Attapulgite clay	88.2%

E and F. Dilution of formulations for use and rate and method of application

There are several dilutions of formulations recommended for use. Many of these dilutions are in combination with other chemicals (2,4-dichlorophenoxy acetic acid or 2,4,5-trichlorophenoxy acetic acid). These dilutions, mixtures, recommended target species, rates, and methods of application for each of the four formulations manufactured are listed in the Appendix.

II. Toxicity data on all formulations (Velsicol Chemical Corporation Bulletin 521-2)

A. Safety data

1. Acute mammalian studies

a. Oral

Oral LD_{50} (acid) - rats: 2900 ± 800 mg/kg
Oral LD_{50} (DMA) - on the following:

Rat	1028 mg/kg
Guinea pig	566 mg/kg
Rabbit	566 mg/kg

b. Dermal

The dimethylamine salt of dicamba administered undiluted to the skin of rabbits and rats produced a very mild irritation when administered daily for 2 weeks. When diluted 1:40 in water, no irritation was observed even after 30 days. There was no evidence of systemic toxicity from percutaneous absorbtion.

c. Inhalation

No evidence of toxicity due to inhalation has been noted. Proper care should be used when applying the herbicide, especially when using a granular form.

d. Eye and skin irritation

Mild irritation was produced on the skin of rats and rabbits if dicamba was applied daily in undiluted strength for 2 weeks. A 0.1 ml. aqueous solution of the DMA salt of dicamba produced no injury when applied undiluted to

the cornea or iris; there was only a low grade irritation which disappeared rapidly. The compound caused no irritation or injury when administered to eyes as a 2 percent or a .2 percent aqueous solution, either as single doses or as repeated doses over a period of a week.

2. Subacute studies

a. Oral

Dicamba was fed for 13 weeks to male and female rats at the rate of 100, 500, 800, and 1000 ppm of the diet. Food consumption and growth rate remained normal, no deaths occurred, and pathology at the end of 7 weeks was negative. At the end of 13 weeks, there was some liver and kidney pathology at the 800 and 1000 ppm level, but none at or below the 500 ppm levels.

Rats, fed at 5, 50, 100, 250, and 500 ppm of diet and dogs, fed at 5, 25, and 50 ppm of diet, showed no apparent effects after 2 years of continuous feeding.

Lactating dairy cattle were fed dicamba at the rate of 10, 25, and 50 ppm of diet. The milk showed no residue of the chemical. When the dosage was raised to 80 and 400 ppm of the diet, residues not exceeding .15 ppm were detected after 9 days of continuous feeding.

b. Dermal

The dimethylamine salt of dicamba administered undiluted to the skin of rabbits and rats produced a very mild irritation when administered daily for 2 weeks. When diluted 1:40 in water, no irritation was observed even after 30 days. There was no evidence of systemic toxicity from percutaneous absorbtion.

c. Inhalation

No evidence of toxicity due to inhalation has been noted. Proper care should be used when applying the herbicide, especially when using a granular form.

3. Other studies which may be required

a. Neurotoxicity

There were no symptoms of neurotoxicity in any studies.

b. Teratogenicity

Rats on a diet containing 500 ppm dicamba for 3 or 4 months did not produce evidence of teratology over a three generation study.

c. Effects on reproduction

Rats on a diet containing 500 ppm dicamba for 3 or 4 months did not show change in reproductive capacity in either parents or offspring.

d. Synergism

There were no synergistic effects in the studies of rats feeding on a diet of 500 ppm dicamba.

e. Potentiation

No evidence of potentiation in any studies of dicamba.

f. Metabolism

Metabolism of rats was not affected by diets of 5, 50, 100, 250, or 500 ppm dicamba over a 2-year period. Dogs on diets of 5, 25, and 50 ppm dicamba showed no effects after 2 years on the diet.

g. Avian and fish toxicity

LD_{50} toxicity of dicamba is set at 673 mg/kg for domestic hens and at 800 mg/kg for pheasants.

LC_{50} toxicity of dicamba on rainbow trout at 24 and 48 hours was 35,000; and at 96 hours, 28,000 micrograms per liter of water. For bluegills at 24 hours, the LC_{50} was 130,000 micrograms; and at 96 hours, 23,000 micrograms per liter. Thus, the concentration which would kill 50 percent of the fish of both species at 96 hours ranges between 23 and 130 ppm of dicamba.

A study on small carp showed that at 24 hours, the LC_{50} for the DMA salt formulation was 659 ppm and at 48 hours, 465 ppm.

The median tolerance limits for juvenile coho salmon exposed to dicamba were 151 and 121 ppm active ingredient for 24 and 48 hours, respectively.

B. Physical-chemical properties

1. Boiling point

The melting point for dicamba is 114 to 116°C.

2. Flash point

Nonflammable

3. Physical state

a. Reference grade - White crystalline solid.

b. Technical grade - Brown crystalline solid.

4. Density

Mollecular weight - 221.05

5. Vapor pressure

3.75 X 10^{-3}mm. Hq. at 100°C.

6. Solubility

Solvent (at 25°C.)	Dicamba gm/100 ml	DMA salt of dicamba (gm acid equivalent per 100 ml)
Water	0.45	72
HAN	5.2	-
Xylene	7.8	-
Ethanol	92.2	-

7. Stability

Stable toward oxidation and hydrolysis under conditions of normal use. Resistant to acid and strong alkali.

III. Efficacy data under field and laboratory conditions. (Velsicol Chemical Corporation newsletter Vol. 1, #2, and Velsicol Chemical Corporation Bull. 521.2.)

A. Effectiveness for intended purposes when used as directed. Dicamba is apparently effective when used as directed on certain plant species. (See I - E and F.) The effectiveness may be less than with other herbicides and is affected by variations in soil, climate, and other variables. Potential users should check with local Agricultural Experiment Stations concerning individual species and/or soils. If local information is not available, a small field

trial may be advisable before investing large sums of money in treatment (Brady, Peevy, and Prine and Starr).

B. Phytotoxicity

Dicamba is readily absorbed through both roots and leaves. Although the mode of action of growth regulator-type compounds has not been fully elucidated, death of susceptible plants treated with dicamba is probably influenced by disruption of normal metabolic and growth activities.

Chlorobenzoic acids as a group have the ability to modify the transport of IAA (Brian). Foy and Penner found that dicamba inhibited the tricarboxylic acid cycle substrate oxidation by mitochondrial fractions isolated from etiolated cucumber cotyledons. Phloem, cambium, and associated parenchyma near the nodes of alligatorweed (Alternanthera sp.) plants were disrupted by treatment with dicamba. Van Overbeek has stated that chlorinated benzoic acids, which have auxin activity, act fundamentally in the same manner as 2,4-D.

C. Translocation with plant or animal treated

Dicamba is absorbed by both roots and leaves. Once inside the plant, the material is translocated in both the xylem and phloem (Cain, Hurtt and Foy, Linder et.al., Leonard et.al., Chang and Vanden Born, and Hall and Brady).

When ingested by dogs, dicamba was rapidly excreted in the urine. About 12 percent of the dose was excreted in conjunction with glycine and the remainder excreted unchanged.

D. Persistence in soil, water, or plants

Harris, Boppart, Markland, Freisen, and Weber and Best have all shown that dicamba is relatively easy to leach from surface layers of soil. Comparitively, dicamba is considered one of the most mobile of the herbicides after it enters the soil.

Sheets et.al., Burnside and Lavy, Chirchrillo, and Velsicol Chemical Corporation have studied degradation of dicamba in the soil. They found that degradation by chemical and/or microbial action was most rapid when soils were at or near 80 percent field capacity and at 25° to 35°C. Under these conditions, breakdown of the chemical was complete within a time frame of 1 to 2 months. The rate of biodegradation increases with temperature; reaching maximum at about 28° to 35°C. At somewhere near 50 percent moisture (by weight), biodegradation reaches maximum and then declines with increasing moisture. These temperatures and moisture contents are conducive to bacterial action. Audus and Cain found

that Bacillus cereus var. mycoides was capable of decarboxylating dicamba and stated that this bacterium is a common organism, found widely distributed in the soils. Studies by Velsicol Chemical Corporation found that dicamba rapidly was broken down or leached from the deeper layers of soil. Within 10 months after application, dicamba applied at up to 6 pounds per acre had disappeared from both the 0-12" and 12-24" depths. Thirty-two inches of rain had fallen during the 10-month period.

There is some evidence that dicamba may be broken down by photo-decomposition (Velsicol Development Newsletter Vol. 1, #2). Since the dimethylamine salt of the acid is quite soluble in water, photo-decomposition might be one of the few ways breakdown occurs in water. Uptake by stream or pond vegetation and ultimate metabolism by the plants would also contribute to clearing water of the chemical. Precautions should be taken to avoid contamination of waterways, ponds, or lakes.

Dicamba is not too persistent in plant tissues. Dissipation can occur by metabolism within the plant; exudation from the roots; and loss from the leaf surface by washing, photo-decomposition, or chemical decomposition (Velsicol Development Newsletter Vol. 1, #2). Malina found that dicamba and its metabolites (5-hydroxy-2 methoxy-3,6-dichlorobenzoic acid and 3,6-dichlorosalicylic acid) were dissipated rapidly from bluegrass and burmudagrass as shown in the following:

Period after treatment (days)	Dicamba (ppm)			5-hydroxy-2 methoxy-3,6-dichlorobenzoic acid (ppm)			3,6dichloro-salicylic acid (ppm)
	2 lb.	5 lb.	10 lb.	2 lb.	5 lb.	10 lb.	
7	51.1	86.2	250.0	33.6	19.3	135.0	negligible (less
14	24.4	51.8	96.0	14.5	34.7	33.6	than 0.05 ppm)
30	6.7	15.9	21.7	11.9	32.0	42.2	amounts in all
60	4.0	4.5	12.5	9.7	11.9	25.3	cases

Morton et.al. found similar dissipation patterns from green tissues of silver beardgrass, little bluestem, dallisgrass, and sideoats grama. It should be noted that both metabolites of dicamba are of low order toxicity. Both metabolites are also herbicidally inactive.

E. Compatibility with other chemicals

The DMA salt of dicamba is compatible with most common organic pesticides as well as nitrogen containing fertilizer solutions

(Velsicol Chemical Corporation Bull. 07-151-501). Precipitation of the free acid from water may occur when this formulation is combined with lime-sulfur, heavy metal salts, or strongly acidic solutions or materials. Compatible salt formulations containing 3.0 pounds dicamba acid equivalent per gallon plus 3.0 pounds 2,4-D acid equivalent per gallon can be diluted in relatively hard water without formation of sediments or precipitates. Compatibility with salt formulations of MCPA is also excellent.

IV. Environmental impact

A. Effects of pesticide on non-target organisms

Like most auxin related herbicides, dicamba has a detrimental effect on plants. Sensitivity of plants varies considerably. Patric and Campbell categorized plants in West Virginia into three susceptibility classes as follows:

Least Susceptible

American beech	Chestnut oak
Fern	Grasses
Hickory	Sedge
Striped maple	Sugar maple
White ash	Witch hazel

Intermediate in Susceptibility

Blackberry	Black birch
Black cherry	Black gum
Chestnut	Cucumber tree
Deertongue grass	Dock
Fireweed	Flowering dogwood
Loosestrife	Red maple
Red oak	Sassafras
Serviceberry	Sourwood
Violet	

Most Susceptible

American elder	Azalea
Bindweed	Black locust
Blueberry	May apple
Mulkin	Nettle
Pin cherry	Pokeweed
Puckley ash	Sheep sorrel
Smartweed	Staghorn sumac
Teaberry	Twisted stalk

They also found that plants were less responsive to pelleted (granules) formulations than to liquid formulations sprayed directly on the foliage. Application of the pelleted formulations were most effective when applied during rapid growth (high moisture content) of the plants.

The effect dicamba might have on beneficial insects or aquatic insects is not documented. However, the chemical's relatively high LD_{50} for several animal species (Section II-A) indicates that mortality due to the chemical should be low or non-existant providing adequate care is taken in application. If the insect is feeding on the more susceptible plants of the area, its food supply would be limited by the plant kill. The insect would then have to move out of the treated area or adjust to some other source of food.

The direct effects of dicamba on fish, birds, wildlife, humans, and domestic animals are shown in Section II-A. Providing adequate controls are maintained, there should be small chance for animals to receive a lethal dose due to treatment. However, the food supply of many wildlife species may be restricted following an application of the herbicide. Animals dependent on the more susceptible species will be forced to leave the area.

The LD_{50} levels for the various animals, fowl, and fish, were established by feeding trials which lasted up to 2 years (see Section II-A).

B. Residues

Studies of the dissipation of dicamba in grass and small grains showed that disruption within living plant tissue was generally logarithmic with time (Velsicol Chemical Corporation Bull. 07-151-501). Application of .5 lb./acre dicamba on wheat (5 leaf stage) showed that residues declined from 63 ppm on day of application to zero 28 days after treatment. In corn, no dicamba residues were detected at ensilage stage when 1 lb/acre was applied preemergence. Postemergence, .25 lb./acre applied up to the time corn was 36 inches tall yielded no residues at ensilage time. The method used on residue analysis was the electron capture gas chromotographic method described by Smith et.al.

Dicamba is evidently excreted rapidly from mammals which may have ingested the chemical. There is no evidence that the chemical is stored and retained by specific organs. Being highly water soluble (in the DMA formulation), the chemical would tend to move through the body rapidly. This proved to be true in a feeding study on dogs (Velsicol Chemical Corporation Bulletin 07-151-501). Dicamba was rapidly excreted in the urine. About

12 percent was excreted in conjugation with glycine and the remainder was unchaged.

Dicamba tends not to remain as a residue in either plants, soil, or water (See III-D).

APPENDIX

This information is taken directly from Velsicol Chemical Corporation Bulletin 07-001-501 and Velsicol Chemical Corporation Pamphlet "Banvel Industrial Herbicides."

1. Banvel Herbicide (Water Soluble) - U.S.D.A. Reg. No. 876-25

FIELD CORN POST-EMERGENCE

USE	WEED	DOSAGE/ACRE	APPLICATION
Field Corn (not registered for use on sweetcorn or popcorn)	Smartweed Canada thistle Cocklebur Pigweeds Lambsquarter Ragweed Mustard Sunflower Velvetleaf Pepperweed Waterhemp Common morning glory Spanish nettle Poorjoe Prostrate Spurge Annual Clover and any other annual broadleaf weeds	**Broadcast** .25 to .50 Pint (2-4 oz. dicamba acid equivalent) 1 gallon treats 16 to 32 acres. **Band** 12" band in 40" row .075 to .150 Pint (.6-1.2 oz. dicamba acid equivalent) 1 gallon treats 53.3 to 106.7 acres. 12" band in 30" row .1 to .2 Pint (.8 to 1.6 oz. dicamba acid equivalent) 1 gallon treats 40 to 80 acres. 12" band in 20" row .15 to .30 Pint (1.2 to 2.4 oz. dicamba acid equivalent) 1 gallon treats 26.7 to 53.3 acres.	BANVEL may be applied over the top of field corn until corn is 36 inches tall or until 15 days before tassel emergence, whichever occurs first. Do not apply BANVEL after this height or growth stage. It is not necessary to use drop nozzles when applying BANVEL alone. Banvel at ¼ pint (2 oz. dicamba acid equivalent) may be tank mixed with 4 to 8 oz. active ingredient of 2,4-D amine for broader spectrum weed control. When 2,4-D is tank mixed with Banvel, drop nozzles are to be used to direct spray to base of corn plant after corn is 8 inches tall. Do not make over one post-emergence application per season.

RATE TO USE

Weeds are easier to kill when they are small and it is suggested that the lower rate of BANVEL be used when weeds are less than 2 inches tall, and the higher rate be used when weeds are over 12 inches tall. Some older weeds are harder to kill and will be suppressed with BANVEL.

FIELD CORN POST-EMERGENCE

BANVEL AND ATRAZINE TANK MIX

CROP	USE	DOSAGE/ACRE	USE DIRECTIONS FOR BANVEL + ATRAZINE TANK MIX
Field Corn	**Grass** Foxtail (giant yellow, green) Crabgrass Barnyardgrass and other annual grass weeds **Broadcast** Smartweed Canada Thistle Pigweed Lambsquarter Ragweed (common, giant) Mustard Velvetleaf Pepperweed Morning glory, common Spanish Nettle Poorjoe Spurge, prostrate Clover, annual Sowthistle Horsenettle Horseweed and other annual broadleaf weeds	**Broadcast** 0.5 pint BANVEL (4 oz. dicamba acid equivalent) plus 1.25 to 2.0 lbs. Atrazine 80 W (1.0 to 1.5 lbs. active ingredient) 1 gallon treats 16 acres **Band** 12" band in 40" row 0.15 pint BANVEL plus .4 to .6 lbs. Atrazine 80 W 1 gallon BANVEL treats 53 acres 12" band in 30" row 0.2 pint BANVEL plus .5 to .8 lbs. Atrazine 80 W 1 gallon BANVEL treats 40 acres 12" band in 20" row 0.3 pint BANVEL plus 0.7 to 1.2 lbs. Atrazine 80 W 1 gallon treats 27 acres	For control of grass and broadleaf weeds, tank mix BANVEL plus Atrazine and make application up to 3 weeks after planting and before grass reaches 1½ inches tall. It may be necessary to cultivate at lay by time to remove surviving weeds or to give soil aeration. Consult the Atrazine 80 W label concerning instruction on method of application and use precautions for Atrazine.

SMALL GRAINS (not underseeded to legumes)

USE	WEED	DOSAGE/ACRE	APPLICATION
Spring Seeded Wheat and Oats	Wild buckwheat Smartweed	.25 Pint (2 oz. dicamba acid equivalent)	Apply at 2 to 5 leaf stage of wheat or oats. May be tank mixed with 4-6 oz. per acre of MCPA or 2,4-D
Spring Seeded Barley (Montana and North Dakota Only)	Wild buckwheat Smartweed	.19 Pint (1.5 oz. dicamba acid equivalent	Apply at 2 to 3 leaf stage of barley. May be tank mixed with 4-6 oz. per acre of MCPA at the 2-3 leaf stage of barley or with 4 oz. per acre of 2,4-D at the 5 leaf stage. Apply only one application per season and do not use higher rate than recommended.
Fall Seeded Barley, Oats and Wheat	Dog fennels (mayweed and corn chamomile) Corn cockle Cow cockle Knawel (German moss)	.25 to .5 Pint (2 to 4 oz. dicamba acid equivalent)	For the suppression or control of weeds, make application immediately after winter dormancy and before grain begins to joint. May be tank mixed with 4-6 oz. per acre of MCPA or 2,4-D.
Fall Seeded Wheat	Fiddleneck Gromwells	.25 to .5 Pint (2 to 4 oz. dicamba acid equivalent) plus .5 to .75 lbs. active ingredient 2,4-D LV ester	Make application immediately after winter dormancy and before wheat beings to join. BANVEL and 2,4-D LV ester to be tank mixed

GRAIN SORGHUM—POST-EMERGENCE

Sorghum (Grain)	Carelessweed (pigweed) Sunflower Lambsquarter Purslane Cocklebur Annual Morning Glory and other annual Broadleaf Weeds	Broadcast .5 Pint (4 oz. dicamba acid equivalent) 1 gallon treats 16 acres Band 20 Inch Band in 40 Inch Row .25 Pint (2 oz. dicamba acid equivalent) 1 gallon treats 32 acres 16 Inch Band in 40 Inch Row .2 Pint (1.6 oz. dicamba acid equivalent) 1 gallon treats 40 acres 12 Inch Band in 40 Inch Row .14 Pint (1.2 oz. dicamba acid treatment) 1 gallon treats 56 acres	BANVEL is to be applied as a post-emergence treatment. For most effective weed control, apply when weeds are small. As weeds become larger they are harder to kill but will be suppressed with BANVEL. BANVEL is to be applied from 10 days after emergence of the grain sorghum from the ground until 25 days after emergence from the ground. Do not apply later than 25 days after emergence of the sorghum from the ground. BANVEL may be applied over the top of sorghum or as a directed application. BANVEL may be used on both irrigated and non-irrigated grain sorghum. Make no more than one application per season. Mix proper amount of chemical with 10 to 25 gallons of water per acre. Do not apply BANVEL to sorghum grown for seed production. Under certain conditions, sorghum may show temporary effects from treatment such as onion leafing or flattening of the plants, but within 10 to 14 days affected plants will recover. See IMPORTANT grazing statement (Page 1) for limitations on grazing and feeding of treated sorghum

GRAIN SORGHUM—HARVEST AID TREATMENT

USE	WEED	DOSAGE/ACRE	APPLICATION
Sorghum, Grain	Careleasweed (pigweed) Lambsquarter Kochia Sunflower Cocklebur Morning glory, annual and other annual broadleaf weeds	**Broadcast** .50 Pint BANVEL (4 oz. dicamba acid equivalent) 1 gallon BANVEL treats 16 acres	Harvest-aid treatment is limited to Texas and Oklahoma and is limited to a single application per crop season. Do not use BANVEL for harvest-aid treatment if BANVEL has been applied earlier that season. For suppression and retardation of susceptible weed, make application from the soft dough stage of the sorghum until 30 days prior to harvest. BANVEL may be applied over the top of sorghum or as a directed application. BANVEL may be used on both irrigated and non-irrigated sorghum. Mix proper amount of chemical with 10 to 25 gallons of water per acre.

GRASS SEED PRODUCTION

For establishment of perennial grasses including bluegrass, lawn-type fescues and other special grasses or Established perennial grasses grown for seed.	Sheep sorrel (red sorrel)	.5 to 1 Pint (4 to 8 oz. dicamba acid equivalent)	For established perennial grasses make application between November 15 and April 1 or prior to boot stage. For new seeding make application to foliage in spring after the seed crop has 3 to 5 leaves. Use sufficient water to give complete coverage (3 to 40 gallons per acre).
	Nightflowering catchfly White cockle Alfalfa	.5 to 1 Pint (4 to 8 oz. dicamba acid equivalent)	For established perennial grasses make application to foliage in spring when seed crop is 2 to 4 inches high. For new seeding see sheep sorrell control directions above.
	Bladder campion Chickweeds (common, mouseear) Stitchwort Clover Curly dock Cow cockle Dog fennels (mayweed and corn chamomile) Knotweed Top growth control of field bindweed, Russian knapweed and Canada thistle	**Established Grass** 1 to 2 Pints (.5 to 1.0 lb. dicamba acid equivalent) **New Seeding** .5 to 1 Pint (4 to 8 oz. dicamba acid equivalent)	For established perennial grasses make application to foliage in spring. For new seeding see sheep sorrell control directions above.
	Downy bromegrass (cheatgrass) Rattail fescue Ripgut brome	2 to 4 quarts (2 to 4 lbs. dicamba acid equivalent)	Make application in fall after harvest and burning and within 3 to 14 days after first irrigation and before weed has more than two leaves.

SPOT APPLICATIONS ONLY OF PERENNIAL BROADLEAF WEEDS IN CROPLAND ROTATED TO WHEAT

LOCATION	WEED	DOSAGE/ACRE	APPLICATION
Idaho Montana Nevada Oregon Utah Washington	Canada thistle, Field bindweed (morningglory), Russian knapweed, Leafy spurge, Tansy ragwort. Black knapweed, Curly dock, Bitter dock	4-6 quarts per acre (4-6 lbs. dicamba acid equivalent)	Spot application may be made to fallow land, wheat stubble, or land to be rotated to wheat. Application can be made in mid-summer to fall of year when weeds are actively growing. WHEAT may be planted one month after application. BANVEL applied at rates of 6 lbs. per acre (dicamba acid equivalent) may cause some wheat injury. See note below.*
Colorado Kansas Nebraska North Dakota South Dakota Wyoming	Canada thistle, Field bindweed, Russian knapweed Leafy spurge	1-2 quarts per acre (1-2 lbs. dicamba acid equivalent)	Spot application may be made to fallow land, wheat stubble or land to be rotated to WHEAT. Application should be made in fall of year when weeds are actively growing. Treatment can be made within 90 days prior to planting or after planting, but before wheat emerges. See note below.*

*Note: In most cases these above treatments will not kill perennial weed seedlings which germinate from seed one or two years after treatment. Once the effect of the chemical has been lost, a follow-up program for seedling control or other cultural practices should be instituted.

PASTURE AND RANGELAND GRASSES AND NON-CROPLAND

USE	WEED	DOSAGE/ACRE	APPLICATION
Pasture and Rangeland Grasses and Non-cropland areas such as fencerows, roadways, wasteland and similar areas	Blood weed Wild buckwheat Annual clover Hubam clover Cowcockle Corn cockle Cocklebur Dogfennels (mayweed, corn chamomile) Knawel (German moss) Knotweed Lambsquarter Mustard Field pennycress Redroot pigweed Tumble pigweed Poorjoe Common ragweed Rabbit brush Sheep sorrel (red sorrel) Smartweed Spanish nettle Spikeweed Prostrate spurge Sunflower Waterhemp	.5 Pint (4 oz. dicamba acid equivalent) For spot treatment mix 0.3 teaspoon BANVEL® Herbicide with 1 gallon water to treat 1 square rod (272 square feet)	For control or suppression of listed weeds, apply BANVEL® when weeds are actively growing. For ground equipment use 10 to 20 gallons of water per acre when treating annual broadleaf weeds and for top growth control of perennial broad leaf weeds. For maximum control of perennial broadleaf weeds use up to 100 gallons or more of water per acre
	Bladder campion Buffalobur Burclover Chickweed Chicory Croton (goatweed) Curly dock Kochia Annual morning-glory Puncturevine Tansy ragwort (rosette stage) Giant ragweed Rattlebush Sesbania Shepherdspurse Teasel Velvetleaf Wormwood	1 Pint (8 oz. dicamba acid equivalent) For spot treatment mix 0.6 teaspoon BANVEL® Herbicide with 1 gallon water to treat 1 square rod (272 square feet)	Rates of BANVEL® in excess of 4 pounds per acre dicamba acid equivalent may cause temporary injury to sensitive grass species. For waiting period between treatment and grazing or harvest of treated grass see IMPORTANT section.

PASTURE AND RANGELAND GRASSES AND NON-CROPLAND (CONT.)

USE	WEED	DOSAGE/ACRE	APPLICATION
Pasture and Rangeland Grasses and Non-cropland areas such as fencerows, roadways, wasteland and similar areas	Top Growth Control: Canada thistle Russian thistle Field bindweed Black knapweed Leafy spurge Perennial sow thistle and other perennial broadleaf weeds	1 Pint (8 oz. dicamba acid equivalent) For spot treatment mix 0.6 teaspoon BANVEL® Herbicide with 1 gallon water to treat 1 square rod (272 square feet)	Rates of BANVEL® in excess of 4 pounds per acre dicamba acid equivalent may cause temporary injury to sensitive grass species. For waiting period between treatment and grazing or harvest of treated grass see IMPORTANT section.
	Spiny aster Slender aster Balloonvine Clover Dwarf mallow Wild garlic Goldenrod Diffuse knapweed Spotted knapweed Wild onion Povertyweed Perennial ragweed Small leaf sida Rough sumpweed Tarbush Sowthistle Tievine Water primrose	1 Quart (1 pound dicamba acid equivalent) For spot treatment mix 1.2 teaspoons BANVEL® Herbicide with 1 gallon water to treat 1 square rod (272 square feet)	
	Blueweed Buckrush Wild carrot Cottonwood (seedlings) Creosotebush Evening primrose Groundsel Spotted knapweed Lote Mesquite Western whorled milkweed Climbing milkweed Stinging nettle Silverleaf nightshade Pepperweed (tall whitetop) Pingue Poison ivy Bur ragweed Tansy ragwort (mature stage) Redvine Sagebrush Perennial smartweed Snakeweed Wood sorrel Musk thistle Trumpet creeper Yarrow Yaupon	2 Quarts (2 pounds dicamba acid equivalent) For spot treatment mix .75 tablespoon BANVEL® Herbicide with 1 gallon water to treat 1 square rod (272 square feet)	Rates of BANVEL® in excess of 4 pounds per acre dicamba acid equivalent may cause temporary injury to sensitive grass species. For waiting period between treatment and grazing or harvest of treated grass see IMPORTANT section.

PASTURE AND RANGELAND GRASSES AND NON-CROPLAND (CONT.)

USE	WEED	DOSAGE/ACRE	APPLICATION
Pasture and Rangeland Grasses and Non-Cropland areas such as fencerows, roadways, wasteland and similar areas	Bedstraw Field bindweed Blackberry Bluebell Bracken fern Prickly pear (cactus) Hop clover Dewberry Grape Carolina geranium Wild honeysuckle Horsemint Horseweed Huisache Russian knapweed Kudzu Bull nettle Poison oak Running live plantain (turbinella) Pokeweed Leafy spurge Sumac Canada thistle Sowthistle Delmation toadflex Vetch White lupine Wild plum Waterhemlock Willow Yucca	1 to 2 gallons (4 to 8 pounds dicamba acid equivalent) For spot treatment mix 1.5 to 3 tablespoons BANVEL® Herbicide with 1 gallon water to treat 1 square rod (272 square feet)	Rates of BANVEL® in excess of 4 pounds per acre dicamba acid equivalent may cause temporary injury to sensitive grass species. For waiting period between treatment and grazing or harvest of treated grass see IMPORTANT section.
	Bracken fern	1 to 2 gallons (4 to 8 pounds dicamba acid equivalent)	Apply as a pre-emergence application before emergence of the fronds.
	Eastern persimmon	1 to 2 gallons in 100 gallons water (4 to 8 pounds dicamba acid equivalent)	Apply to ground under tree as basal treatment using .13 to .25 pint of spray solution per inch diameter of the plant. May also be used as a stem foliage treatment with sufficient water to give good coverage.

NON-CROPLAND — BRUSH CONTROL

USE	WEED	DOSAGE/ACRE	APPLICATION
Fencerows, Roadways, Utility Rights-of-Way, Wasteland and Similar Non-Cropland	Mixed brush including both deciduous (hardwood) and evergreen species. A partial list of trees controlled by BANVEL + 2,4-D or 2,4,5-T is as follows: ash — persimmon aspen — pine basswood — poplar cedar — sassafras cherry — service berry chinquapin — spicebush cucumber-tree — sour wood gum — sumac dogwood — sycamore elm — thornapple hickory — thornberry hornbeam — willow locust — witch hazel maples — yaupon oak — and others	1.25 Quarts 1.25 Lbs. dicamba acid equivalent) per 100 gallons water plus 2.5 Lbs. active ingredient 2,4-D or 2,4,5-T (amine or L.V. ester) per 100 gallons of water	For broad spectrum brush control, tank mix BANVEL with 2,4-D or 2,4,5-T. Treat all stems and foliage with special emphasis on covering the root crown. For best results apply at the rate of 200 to 300 gallons of pater per acre. Lesser amounts of water may be used but maintain minimum of 2.5 quarts of BANVEL per acre when tank mixed with 2,4-D or 2,4,5-T. Make repeat application when needed.
	Eastern persimmon	1 to 2 gallons in 100 gallons water (4 to 8 lbs. dicamba acid equivalent)	Apply to ground under trees as basal treatment using .13 to .25 pint of spray solution per inch diameter of the plant. May also be used as a stem foliage treatment with sufficient water to give good coverage.

TREE INJECTION

Tree kill by injection	Alder (Red) Ash (White) Aspen Basswood Beech Birch (Yellow, Paper) Dogwood Gum (Sweet, Black) Hickory Huckleberry Maple (Red, Sugar) Oak (Blackjack, Post) Persimmon Pine (White)	Mix 1 part BANVEL Herbicide to 1 part water or use BANVEL Herbicide undiluted Apply .5 to 1.0 milliliter (ml.) per injection. Overlap cuts or space cuts up to 2 inches apart from edge to edge	May be applied anytime during the year. To obtain satisfactory kill the cut must penetrate the bark and the cambium layer (sapwood). Application may be made by special designed injector that meters out desired quantity of chemical or cuts may be made with an axe and chemical applied with an oil can or other suitable applicator. Symptoms of injury will be noted within a few weeks but kill may take several months.
	Oak (Black, Chestnut, Red, White) Pine (Shortleaf)	Mix 1 part BANVEL to 4 parts water. Apply .5 to 1.0 milliliter (ml.) of mix per injection Space cuts up to 3 inches apart from edge to edge	

BANVEL INDUSTRIAL BRUSH AND WEED CONTROL LABEL REGISTRATIONS

USE	WEED/BRUSH	DOSAGE/ACRE	APPLICATION
Rights-of-Way (utility, railroad, highway, pipeline). Non-selective forest brush control, fence-rows, drainage ditch banks, wasteland and similar non-cropland.	Unwanted woody brush including both hardwood and evergreen species. A partial list of trees controlled by BANVEL + 2,4D- is as follows: Alder Ash Aspen Basswood Cedar Cherry Chinquopin Cucumber tree Gum Guava Dogwood Elm Hemlock Hickory Hornbean Locust Maple Oak Persimmon Pine Poplar Sassafras Schinus (Christmasberry) Service berry Spicebush Spruce Sycamore Thornapple Thornberry Willow Yaupon Mesquite Sumac Wild plum Witch Hazel and many other woody plant species	1 quart BANVEL (1.0 lb. dicamba acid equivalent) per 100 gallons water plus 2.0 lbs. active ingredient 2,4-D (amine or L.V. ester) per 100 gallons water	**Hydraulic Spray Application Stem Foliage – High Water Volume** Tank mix BANVEL with 2,4-D and make application after leaves are fully developed until three weeks before frost. Treat all stem and foliage to run-off with special emphasis on covering the root crown. Depending upon height and density of the brush, apply 200 to 300 gallons of spray mix per acre.
		2¼ gallons BANVEL (9.0 lbs. dicamba acid equivalent) per 100 gallons of water plus 18.0 lbs. active ingredient 2,4-D (amine or L.V. ester) per 100 gallons water	**Back Pack Mist Blower Application Basal Stem Foliage – Low Water Application** Tank mix BANVEL with 2,4-D and make application after leaves are fully developed. Treatment may be made up to three weeks of frost. Treat all stem and root crown to run-off. Use mist blower application on brush 6 feet tall or less at the rate of 30 to 35 gallons total spray mix per acre.
		6.0 gallons BANVEL (24 lbs. dicamba acid equivalent) plus 12 gallons (4 lbs. active ingredient/gallon) 2,4-D (amine or L.V. ester) in 82 gallons water (total 100 gallons spray mix)	**Aerial Application – 12 Gallons Spray Mix Per Acre** Tank mix BANVEL + 2,4-D at the given rate when applying *12 gallons of total spray mix per acre.* Treatment may be made from the time the leaves are fully developed until 3 weeks before frost.
		2.5 gallons BANVEL (10 lbs. dicamba acid equivalent) plus 5.0 gallons (4 lbs. active ingredient/gallon) 2,4-D (amine or L.V. ester) in 92.5 gallons water	**Aerial Application – 30 Gallons Spray Mix Per Acre** Tank mix BANVEL + 2,4-D at the given rate when applying *30 gallons of total spray mix per acre.*

BANVEL INDUSTRIAL BRUSH AND WEED CONTROL LABEL REGISTRATIONS (CONT.)

USE	WEED/BRUSH	DOSAGE/ACRE	APPLICATION
Rights-of-Way (Utility, railroad, highway, pipeline). Non-selective forest brush control, fencerows, drainage ditch banks, wasteland and similar non-cropland.	For control of annual and deep rooted perennial broadleaf weeds. A partial list of weeds controlled by BANVEL and BANVEL + 2,4-D mixtures is as follows: Curly dock Field bindweed (morning glory) Leafy spurge Russian Knapweed Canada Thistle Tansey Ragwort Puncture Vine Perennial Ragweed Tievine Milkweed Redvine Dalmatian toadflax and many other perennial broadleaf weeds **Annual Broadleaf Weeds** Wild buckwheat Smartweed Pigweed Lambsquarter Ragweed Mustard Velvetleaf Chickweeds Dogfennels Clover Sheep sorrel Henbit English daisy Purslane Carpetweed Cocklebur Knawel	1.0 pint BANVEL (0.5 lb. dicamba acid equivalent) plus 1.0 to 2.0 lbs. active ingredient 2,4-D (amine or L.V. ester) per 100 gallons water.	For effective broad spectrum control of annual and perennial broadleaf weeds tank mix BANVEL + 2,4-D as directed under rate of application. Make application when weeds are actively growing. Apply at the rate of 100 to 200 gallons of spray mix per acre. If lower volumes of water are used then increase the amount of chemical per 100 gallons of water accordingly. If perennial broadleaf weeds are the predominant weed problem, then use the higher spray rate.
	Bracken fern	1 to 2 gallons (4 to 8 lbs. dicamba acid equivalent) per acre	Apply as a pre-emergence application before emergence of the fronds in sufficient water to give good coverage.

2. Banvel Brush Killer (Oil Soluble)

BANVEL® 4-O.S. is to be tank-mixed with 2,4-D or 2,4,5-T ester which is soluble in oil. This combination is to be used with diesel oil or fuel oil. DO NOT USE WITH WATER.

Use *BANVEL® 4-O.S.* plus 2,4-D or 2,4,5-T ester to control unwanted woody plants along utility, railroad, highway and pipeline rights-of-way; for nonselective forest brush control; and brush control in wasteland and similar noncropland areas.

BANVEL® 4-O.S. plus 2,4-D or 2,4,5-T ester controls both hardwood and evergreen species, such as alder, apple, ash, beech, birch, cascara, cedar, cherry, dogwood, elderberry, elm, fir, grape, hemlock, hickory, hornbeam, locust, maple, oak, pine, poplar, sassafras, spruce, sumac, walnut, willow and other woody plant species.

DORMANT STEM BROADCAST	BASAL BARK TREATMENT
Treat any time brush is dormant and most of the foliage has dropped off. Thoroughly wet the entire brush or tree to runoff. For root-sucking species, put special emphasis on covering the root crown.	Spray the basal parts of the brush and tree trunk from the ground line up to a height of 1½ to 2 feet. Spray until runoff with special emphasis on covering the root crown. Thorough wetting of the indicated area is needed to achieve good control. Treatment may be made at any time during the year, including the winter (except when snow or water prevents spraying to the ground line).
GROUND APPLICATION For hydraulic spray application—tank-mix 1-3 quarts (1-3 lbs. dicamba acid equivalent) of BANVEL® 4-O.S. with 2-6 lbs. acid equivalent of 2,4-D or 2,4,5-T oil soluble ester in sufficient oil to make 100 gallons of spray mixture. Apply at the rate of 100 gallons of spray mix per acre.	For hydraulic spray application, tank-mix 1-3 quarts (1-3 lbs. dicamba acid equivalent) of BANVEL® 4-O.S. with 2-6 lbs. acid equivalent of either 2,4-D or 2,4,5-T oil soluble ester in sufficient oil to make 100 gallons of spray mixture. Use 100 gallons of spray mixture per acre.
For back-pack mist blower application, tank-mix 2-4 gallons (8-16 pounds dicamba acid equivalent) of BANVEL® 4-O.S. with 16-32 pounds acid equivalent of either 2,4-D or 2,4,5-T oil soluble ester in sufficient oil to make 100 gallons of spray mixture. Apply at the rate of 30 gallons of spray mixture per acre.	For back-pack mist blower application, tank-mix 2-4 gallons (8-16 lbs. dicamba acid equivalent) of BANVEL® 4-O.S. with 16-32 lbs. of either 2,4-D or 2,4,5-T oil soluble ester in sufficient oil to make 100 gallons of spray mixture. Apply at the rate of 30 gallons of spray mixture per acre.
AERIAL APPLICATION (Western Oregon and Washington only) Tank-mix 1 quart (1 lb. dicamba acid equivalent) of BANVEL® 4-O.S. with 4 lbs. acid equivalent of 2,4-D or 2 lbs. acid equivalent of 2,4,5-T oil soluble ester in sufficient oil to make 10-20 gallons of solution. Apply at the rate of 10-20 gallons per acre.	

3. Banvel 5G Granules - U.S.D.A. Reg. No. 876-103

USE	WEED/BRUSH	DOSAGE	APPLICATION
Pasture, rangeland, and non-cropland areas such as fencerows, roadways, wasteland and similar areas	Eastern Persimmon	Use 2 level teaspoonsful of BANVEL 5% Granules per inch diameter of the trunk of the plant. (Example: Use 6 teaspoonsful for a tree with a trunk 3 inches in diameter)	Scatter the granules evenly on the ground within 6 inches of the trunk. Apply BANVEL granules any time after buds start to open and before the leaves and branches stop growing in the summer.
	Creosotebush Tarbush	Use 2 heaping tablespoonsful of BANVEL 5% Granules per 4 feet diameter of canopy	Make application just prior to or in the early part of the rainy season. Scatter the granules uniformly under the canopy of the shrub.
	Salt Cedar	Use 100 to 200 pounds of BANVEL 5% Granules per acre (5 to 10 lbs. dicamba acid equivalent)	Make application just prior to or in the early part of the rainy season. Apply BANVEL granules uniformly over the area to be treated.
	Canada Thistle Field Bindweed (Morning glory) Russian knapweed Leafy spurge Bur ragweed Skeletonweed	Apply at the rate of 80 to 160 lbs. BANVEL 5% Granules (4 to 8 lbs. dicamba acid equivalent) per acre. For spot treatment apply 0.5 to 1.0 lbs. BANVEL 5% Granules per sq. rod (272 sq. ft.)	For best results, apply BANVEL granules uniformly when plants are actively growing. This would normally be in the spring or fall when plants are putting out new growth.
	Bracken fern	Apply at the rate of 120 to 160 lbs. BANVEL 5% Granules (6 to 8 lbs. dicamba acid equivalent) per acre. For spot treatment apply .75 to 1.0 lb. BANVEL 5% Granules per sq. rod (272 sq. ft.)	Apply granules uniformly as a preemergence application before emergence of the fronds.
	Artichoke thistle	Apply at the rate of 20 to 40 lbs. BANVEL 5% Granules (1 to 2 lbs. dicamba acid equivalent) per acre. For spot treatment apply 2 to 4 oz. BANVEL 5% Granules per sq. rod (272 sq. ft.)	Make uniform application of BANVEL granules when plants are actively growing.

4. Banvel 10G Granules contain twice the active ingredient as Banvel 5G Granules. Target species for both formulations are the same but the 10G Granules are especially useful for spot treatment of areas where low densities of target species occur. This formulation is especially useful on eastern persimmon, creosotebush, tarbush, and salt cedar. One-half the suggested dosage for 5G Granules should be sufficient.

LITERATURE

Audus, L. J. 1964. The physiology and biochemistry of herbicides. Academic Press. pp. 104-206.

Boppart, E. A. 1966. Chemical leaching and bioassay of Banvel D granules. Biological Research Section, Herbicide Report 47-H-66. Velsicol Chemical Report.

Brady, Homer A. 1971. Other brush-control sprays compared to 2,4,5-T ester. Southern Weed Science Society. Proc. of. 24:251-254.

Brian, R. D. 1964. The classification of herbicides and type of toxicity. In: The physiology and biochemistry of herbicides. Edited by: L. J. Audus. Academic Press. 555 pp.

Broadhurst, N. A., M. L. Montgomery and V. H. Freed. 1966. Metabolism of 2-methoxy-3,6-dichlorobenzoic acid (dicamba) by wheat and bluegrass plants. J. Agr. Food Chem. 14:585-588.

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

Cain, P. S. 1966. An investigation of the herbicidal activity of 2-methoxy-3,6-dichlorobenzoic acid. Thesis, Ph.D. U. of Ill., Agronomy, 131 pp.

Chang, F. Y. and W. H. Vanden Born. 1968. Translocation of dicamba in Canada thistle. Weeds. 16:176-181.

Chirchrillo, M. T. 1968. Biodegradation of Banvel D under varying conditions of temperature and moisture. Microbiology Lab. Report No. 15. Velsicol Chemical Corp. (Unpublished).

Corbin, F. T. 1967. Influence of pH on the detoxification of herbicides in soil. Southern Weed Conference. Proc. of. 20:394.

Corbin, F. T. and R. P. Upchurch. 1967. Influence of pH on detoxification of herbicides in soil. Weeds. 15:370-377.

Friesen, H. A. 1965. The movement and persistence of dicamba in soil. Weeds. 13:30-33.

Foy, D. L. and D. Penner. 1965. Effect of inhibitors and herbicides on tricarboxycylic acid cycle substrate oxidation by isolated cucumber mitochondria. Weeds. 13:226-231.

Hall, Oscar and Homer A. Brady. 1971. Mixing herbicides alters their behavior in woody plants. Southern Weed Science Society. Proc. of. 24:255-262.

Harris, C. I. 1963. Movement of dicamba and diphenamid in soils. Weeds. 12:112-115.

Harris, C. I. 1967. Movement of herbicides in soil. Weeds. 15:214-216.

Hurtt, W. and C. L. Foy. 1965. Some factors affecting the excretion of foliarly applied dicamba and picloram from roots of Black Valentine beans. Plant Physiology, Supplement. 40:48.

Leonard, O. A., L. A. Lider and R. K. Glenn. 1966. Absorbtion and translocation of herbicides by Thompson Seedless (Sultanina) Grape, Vitus vinifera L. Weed Res. 637-49.

Linder, P. J., J. W. Mitchell and G. D. Freeman. 1964. Persistence and translocation of exogenous regulating compounds that exude from roots. J. Agr. Food Chem. 12:437-438.

Markland, F. E. 1968. Evaluation of encapsulated granules of Banvel D for leaching characteristics. Biological Res. Section, Herbicide Report 31-H-68. Velsicol Chemical Corp.

Morton, H. L., E. D. Robison and R. E. Meyer. 1967. Persistence of 2,4-D, 2,4,5-T, and dicamba in range forage grasses. Weeds. 15:268-271.

Pate, D. A., H. H. Funderburk, Jr., J. M. Lawrence, and D. E. Davis. 1965. The effect of dichlobenil and dicamba on nodal tissues of alligatorweed. Weeds. 13:208-210.

Patric, James H. and John Campbell. 1970. Some experiences with dacamba in controlling revegetation of deforested land in West Virginia. NE Weed Control Conf. Proc. Vol. 24:61-68.

_____ 1969. A substitute for 2,4,5-T in eastern hardwood sprout and brush control. NE Weed Control Conf. Proc. Vol. 23:320-328.

Peevy, Fred A. 1971. Wide-spaced injections of herbicidal mixtures for controlling weed trees. Southern Weed Science Society. Proc. of. 24:263-267.

Prine, E. Lynn and John W. Starr. 1971. Herbicide control of Japenese honeysuckle in forest stands. Southern Weed Science Society. Proc. of. 24:298-300.

Reinhart, K. G. 1965. Herbicide treatment of watersheds to increase water yield. NE Weed Control Conf. Proc. Vol. 19:546-551.

Sheets, T. J., C. I. Harris, D. D. Kaufman and P. C. Kearney. 1964. Fate of herbicides in soils. NE Weed Control Conf. Proc. Vol. 18:21-31.

Smith, M., H. Suzuki and M. Malina. 1965. Analysis of dicamba in crops and milk, including a rapid cleanup method. Jour. Assn. Official Agr. Chemists. 48:1164.

Technical Services Request F-31. 1965. Soil dissipation study, Banvel D residues. Velsicol Chemical Corp.

Technical Services Request F-59. 1965. Banvel D residues. Velsicol Chemical Corp.

Van Overbeek, J. 1964. Survey of mechanisms in herbicide action. In: The physiology and biochemistry of herbicides. Edited by: L. J. Audus, Academic Press. 555 pp.

Velsicol Chemical Corp. 1971. Banvel herbicides general bulletin. Velsicol Chemical Corp. Bull. 07-151-501:4 pp.

Velsicol Chemical Corp. 1971. Banvel federal label registrations. Velsicol Chemical Corp. Bull. 07-001-501:15 pp.

Velsicol Chemical Corp. 1971. Banvel herbicides for brush and broadleaf weed control. Velsicol Chemical Corp. unnumbered pamphlet. 7 pp.

Wart, D. J. 1964. Effect of herbicides on plant composition and metabolism. In: The physiology and biochemistry of herbicides. Edited by: L. J. Audus, Academic Press. 555 pp.

Weber, J. B. and J. A. Best. 1971. Activity and movement of 13 soil-applied herbicides as influenced by soil reaction. Southern Weed Science Society. Proc. of. 24:403-413.

Weed Science Society of America. 1970. Herbicide handbook of the Weed Science Society of America. WSSA Monograph #3:136-139.

REPORT

ON

BACKGROUND INFORMATION

FOR

MSMA

I. GENERAL INFORMATION

A. Common Name. MSMA.

B. Chemical Name. Monosodium acid methanearsonate or monosodium methanearsonate.

C. Registered Uses. For post-emergent weed control and as a silvicide for control of undesirable conifers and big leaf maple.

D. Formulations Manufactured. (See Table 1 for materials other than silvicides.

1. Silvisar 550 Tree Killer. 6.0 lbs. MSMA per gallon. USDA Reg. No. 6308-58.

2. Vichem 120 Arsonate Silvicide. 6.66 lbs. MSMA per gallon. USDA Reg. No. 2853-39.

3. Glowon Tree Killer. 5.5 lbs. MSMA per gallon. USDA Reg. No. 10592-1.

E. Dilution of Formulation for Use. Use in undiluted form as a silvicide.

F. Rate and Method of Application

1. Silvisar 550 Tree Killer

a. Spaced-Cut Injection with Ansul "Hypo-Hatchet" Injector. This hatchet-like unit cuts and injects in one operation. The injector works by inertia and is calibrated to inject at least one milliliter of chemical per stroke. Rates for this method are:

(1) Conifers and Big Leaf Maple (Growing Season). For trees below 8 inches diameter at breast height (d.b.h.), make one cut per 2 inches of d.b.h. ($4\frac{1}{2}$" spacing between cut edges) at waist height or below. For trees 8 inches d.b.h. and larger, make one cut per 1 inch d.b.h. ($1\frac{1}{2}$" spacing between cut edges).

(2) Conifers (Dormant Season). Make one cut per 1 inch of d.b.h. ($1\frac{1}{2}$" spacing between cut edges) at waist height or below.

(3) Big Leaf Maple (Dormant Season). Make a complete frill at waist height or below (cuts need not be overlapping).

Table 1.--Solutions containing monosodium methanearsonate that are registered for uses other than as a silvicide.

Trade name	Mfg. by	Lbs. MSMA per gal.	USDA or EPA Reg. No.	Use
Ansar 529 H.C.	The Ansul Co.	6.0	6308-29	Selective post-emergent weed control
Ansar 170 H.C.	The Ansul Co.	8.0	6308-60	Selective post-emergent weed control
Phyban H.C.	The Ansul Co.	6.0	6308-37	General post-emergent weed control
Mad	The Ansul Co.	4.0 + 1.0 lb. 2,4-D	6308-64	Selective post-emergent weed control
Broadside	The Ansul Co.	3.0 + 1.25 lbs. cacodylic acid	6308-65	General post-emergent weed control
Monex 3	The Ansul Co.	6.0	6308-72	Selective post-emergent weed control
Arsonate liquid	Diamond Shamrock Chemical Co.	6.6	677-204-AA	Post-emergence weed control in cotton, turf, and non-crop areas
Bueno 6	Diamond Shamrock Chemical Co.	6.0	677-269-AA	Post-emergence weed control in cotton, turf, and non-crop areas
Bueno	Diamond Shamrock Chemical Co.	4.0	677-231-AA	Post-emergence weed control in cotton, turf, and non-crop areas
Daconate 6	Diamond Shamrock Chemical Co.	6.0	677-268-AA	Post-emergence weed control in cotton, turf, and non-crop areas
Daconate	Diamond Shamrock Chemical Co.	4.0	677-199-AA	Post-emergence weed control in cotton, turf, and non-crop areas
WEED-HOE 120	Vineland Chemical Co.	6.66	2853-30	Control of Johnson grass and other weeds in cotton
CHEX-MATE	Vineland Chemical Co.	3.0 + 1.25 lbs. cacodylic acid equiv.	02853-50001 AA, Calif. Reg. No. only	Post-emergence for general weed control

b. Spaced-Cut Application. A hatchet or similar cutting tool can be used to make the cut, and the MSMA added to the cut with a pump-type oil can, plastic squeeze bottle, or other suitable dispenser. Rates for this method are:

(1) Confers and Big Leaf Maple (Growing Season). For trees below 8 inches d.b.h., apply 1 to 2 milliliters of Silvisar 550 Tree Killer per cut per 2 inches of d.b.h. (6" spacing between cut centerlines) at waist height or below. For trees 8 inches d.b.h. and larger, use 1 or 2 milliliters per cut per 1 inch d.b.h. (3" spacing between centerlines).

(2) Conifers (Dormant Season). Apply 1 to 2 milliliters of Silvisar 550 Tree Killer per cut per 1 inch of d.b.h. (3" spacing between cut centerlines).

(3) Big Leaf Maple (Dormant Season). Apply 1 to 2 milliliters of Silvisar 550 Tree Killer per cut in a complete frill at waist height or below. (Cuts need not be overlapping.)

2. Vichem 120 Arsonate Silvicide - Conifers Only. Cut-frills are made with an ax.

a. Growing Season. For control of conifers under 7" d.b.h. with a live crown of less than one-half the total tree height, apply Vichem 120 Silvicide at 1 milliliter (ml) per frill and cut a frill for every 2 inches of tree d.b.h. For larger diameter trees or trees with a full live crown, apply Vichem 120 Silvicide at 1 ml per frill and cut a frill for every 1 inch of tree d.b.h.

b. Dormant Season. For control of conifers under 7" d.b.h. with a live crown of less than one-half the total tree height, apply Vichem 120 Silvicide at 1 ml per frill and cut a frill for every 2 inches of tree d.b.h. For larger diameter trees or trees with a full live crown, apply Vichem 120 Silvicide at 1 to 2 ml per frill and cut almost overlapping frills completely around the circumference of the tree, especially on larger trees.

3. Glowon Tree Killer. Effective on conifers but not on most hardwoods. Apply undiluted into horizontal ax frills cut on the trunk usually at waist height or below. Use the same dosage in dormant season as during growing season. For control of young conifers, less than 6" d.b.h., with a full crown, apply Glowon Tree Killer at 1 ml per frill and cut a frill for every inch of tree d.b.h. For control of young conifers, less than 6" d.b.h., with only half or less of a complete crown, apply Glowon Tree Killer at 1 ml per frill and cut a frill for every 2 inches of tree d.b.h. Large trees with

full crowns require almost overlapping frills for effective control. Do not store in or place in contact with aluminum, copper, or galvanized metal containers.

G. Tolerances in Food or Feed and Other Safety

1. Tolerance as As_2O_3, 0.7 ppm in cottonseed, and 0.9 in cottonseed hulls.

2. Safety Limitations

a. Cotton. Do not graze or feed forage from treated areas to livestock.

b. Drainage Ditch Banks. Do not graze treated areas. Do not contaminate waters used for domestic consumption, or by animals, wildlife, and aquatic life, or for irrigation purposes.

H. Manufacturers or Producers

1. The Ansul Company, Marinette, Wisconsin.

2. Vineland Chemical Company, Vineland, New Jersey.

3. Key Chemicals, Inc., Anacortes, Washington.

II. TOXICITY DATA ON MSMA[1/]

A. Safety Data

1. Acute Mammalian Studies

a. Oral

(1) Acute Oral LD_{50}

(a) Technical Grade Methanearsonic Acid. 92.8% methanearsonic acid; 1.4 g per kg (adult male albino rats); WARF Institute (Wisconsin Alumni Research Foundation, Madison, Wisconsin).

(b) Ansar 170. 51.3% MSMA; 1.8 g per kg (young male and female albino rats); Industrial Bio-Test Laboratories, Northbrook, Illinois.

1/ Except where noted, toxicity data was summarized from a report by The Ansul Company, Marinette, Wisconsin, entitled "Toxicological Data - Methanearsonic Acid and Dimethylarsinic Acid," June 5, 1967, with an addendum dated October 10, 1969.

(c) Ansar 529. 34.8% MSMA; 1.8 g per kg (young male and female albino rats); Industrial Bio-Test Laboratories.

(2) Acute Oral LD_{100}

(a) Ansar 170. 325 mg per kg (dairy calves); E. S. Erwin & Associates, Phoenix, Arizona.

(b) Ansar 529. 400 mg per kg (dairy calves); E. S. Erwin & Associates.

(3) Other Acute Oral Studies

(a) Rodents. Meliere (1969) has measured the acute oral toxicity of methanearsonic acid in male mice and has found that the LD_{20} is 185 mg/kg. He found the LD_{50} of the disodium methanearsonic acid to be greater than 245 mg/kg. In data supplied by the manufacturers and listed in the Suggested Guide for Weed Control (USDA Agricultural Handbook 332, 1967), the acute oral toxicity for rats of the monosodium and the disodium methanearsonic acids is 700 mg/kg for MSMA and 800 to 2,800 for DSMA. These figures are slightly at variance with the toxicity data supplied by The Ansul Chemical Company. It is clear that methanearsonic acid and its salts MSMA and DSMA have about the same acute oral toxicity in rats as cacodylic acid and are less toxic to rats and mice than is sodium arsenite. It also appears that the disodium methanearsonate (DSMA) is much less than the parent acid and the difference is greater than might be predicted on the basis of their arsenic content.

(b) Steers. Dickenson (see Norris 1971) fed a commercial formulation of MSMA to steers. He found lethal effects after 10 mg/kg/day for 10 days. Additional work remains to be completed in Dickenson's study.

b. Estimated Acute Dermal LD_{50} - Ansar 529. 2-4 g per kg (adult male rabbits). The skin irritation tests with methanearsonic acid were conducted in the usual manner using the intact skin of three rabbits and the abraded skin of three rabbits. After 24- and 72-hour exposure to this herbicide, it was found to produce a slight edema and to be mildly irritating. The reactions observed in the highest doses prior to death included general inactivity, loss of appetite, mild sedation, dyspnea, and muscular weakness. At 1,400 mg/kg, the animals which succumbed lived about 24 hours following administration. WARF Institute.

c. Acute Inhalation - Ansar 529. Non-irritating to the respiratory tract (albino rats); WARF Institute.

d. Eye Irritation - Ansar 529. Non-irritating to the eye (adult albino rabbits); WARF Institute.

2. Subacute Studies

a. Oral

(1) Twenty-Four- (24) Hour Foraging on Treated Johnson Grass by Dairy Calves

(a) Ansar 170. Four calves were pastured for 24 hours on 20- by 30-foot (1/70th acre) plots of thick Johnson grass 3 to 4 feet high, previously sprayed to run-off with Ansar 170 at a rate of 1.05 gallons per acre in 70 gallons of water. There was no evidence of unpalatability due to the herbicide. All calves showed a mild diarrhea, which disappeared 48 hours after removal from the plots. No other symptoms appeared for 2 weeks post-treatment. E. S. Erwin & Associates.

(b) Ansar 529. Same test and results as above, but sprayed with Ansar 529 at a rate of 1.75 gallons per acre in 70 gallons of water. E. S. Erwin & Associates.

(2) One-Week Feeding to Dairy Calves - Ansar 529. 529 was fed at 40, 80, and 240 mg/kg, in 8 pounds of supplemental cottonseed meal, to each of two Holstein calves at each level, each day for 1 week. 40 mg/kg - meal consumption dropped to about 4 pounds after 1 day; 80 mg/kg - calves quit feeding on 4th day; 240 mg/kg - calves quit feeding on 3rd day. None of the calves developed diarrhea. All calves recovered rapidly when returned to normal ration at the end of 7 days. E. S. Erwin & Associates.

(3) Lactating Cattle Feeding Study - Pure Methanearsonic Acid. Methanearsonic acid was fed at 0.3, 3.0, and 30 ppm in 5.5 pounds of supplemental cottonseed meal, to each of three lactating cows at each level, daily for 9 weeks. No significant residues in milk and edible tissues. Methanearsonic acid is poorly absorbed in intestinal tract and rapidly excreted in the urine. Dr. S. A. Peoples, Department of Physiological Sciences, University of California, Davis, California.

(4) Ninety- (90) Day Feeding Study in Rats and Dogs - Pure Methanearsonic Acid. Methanearsonic acid was fed at 3, 15, and 30 ppm to dogs, and at 3, 15, 30, and 100 ppm to rats, in the basal ration for 90 days. No-effect level for dogs - 30 ppm; no-effect level for rats - 100 ppm. WARF Institute.

b. Dermal. Considered mildly irritating to the skin (albino rabbits). WARF Institute.

3. Other Studies

a. Carcinogenicity. Arsenic has only been associated with poisoning and was indicated quite early as a carcinogen. More evaluations suggest that the early tests reporting arsenic-induced carcinoma were inadequate. Frost (1970) cites numerous studies which attempted but failed to demonstrate arsenic-induced carcinoma. Cacodylic acid was placed in group c4 by the Secretary's Commission on Pesticides and their relationship to environmental health (Mrak 1969). This group contains pesticides which was judged not positive for carinogenicity in one species (mouse), but current guidelines require negativity in two species. The commission gave this group a moderate priority for testing, but felt no changes in practices in the field were warranted. The similarities in chemical and physical properties of MSMA and cacodylic acid justify extrapolation of data between these two compounds.

S. S. Pinto and B. M. Bennett (1963) believe that it is a mistake to make blanket condemnations of the use of arsenic without first looking at the data. He has reviewed the early literature on human tumors from arsenic and also the recent opinions and interpretations of these early papers. There is reason to believe that the "arsenic tumors" observed in 1820 may have been due to other causes such as selenium poisoning. He reviewed the medical histories and causes of death of the long-term employees of a copper smelting company producing arsenic trioxide. He showed that the workers do excrete high levels of arsenic, but that their incidence of cancer is no greater than for other persons in the State of Washington. He concluded that there is no evidence that exposure of these workers to arsenic trioxide is a cause of systemic cancer in humans. In a sense, this amounts to the use of human guinea pigs for establishing the lack of carcinogenicity of arsenic trioxide.

b. Mutagenicity. Cacodylic acid is a mitotic poison in mammalian organisms. King and Ludford (1960) found that injections in mice produced "profound disturbances of cell division" and it "stimulated mitosis in cells of the crypts of Lieberkuehn" and of transplanted tumors. The significance of this finding in terms of exposure to MSMA and cacodylic acid in the field is not known.

c. Teratogenicity. Cacodylic acid is considered to be a teratogenic agent, producing abnormalities during embryonic development. There are several references to this type of action, although only two examples are quoted. Salzgeber (1955) observed

teratogenic effects in 10-day chick embryo genital organs cultured in vitro and has reported that the greatest damage is to the cortical region. Rostand (1950) has treated tadpoles of Rana temporia with solutions of cacodylic acid for 3 weeks when the hind legs were in the process of development, and abnormalities were observed at 0.10 percent of sodium cacodylate. (This concentration is 100 ppm and is equivalent to 270 lb/acre ft. of water.)

Additional testing, using the techniques reported by Mrak (1970), is needed. Relation of these reports of teratogenic potential and field use of the chemical require further investigation.

d. Avian Toxicity - Chicken Feeding Study - Pure Methanearsonic Acid. Methanearsonic acid was fed at 0.03, 0.3, and 3.0 ppm in basal ration to each of nine Leghorn hens at each level, daily for 4 weeks. No arsenic residues in meat at all levels. Slight arsenic residues in eggs at 3.0 ppm. No pathological evidence of toxicity at any level. Dr. S. A. Peoples.

e. Fish Toxicity

(1) Pure MSMA (no surfactant present). 48-hour LC_{50} - above 1000 ppm (bluegill sunfish). Louisiana Wildlife & Fisheries Commission.

(2) Ansar 529 (surfactant present). 96-hour LC_{50} - 31.1 ppm (goldfish); 96-hour LC_{50} - 13.4 ppm (fathead minnows). Bureau of Sport Fisheries & Wildlife.

(3) Ansar 529 (surfactant present). 96-hour TLm (median tolerance limit) - 300 ppm (bluegill sunfish). Louisiana Wildlife & Fisheries Commission.

B. Physical-Chemical Properties[2/]

1. Boiling point - none
2. Flash point - none
3. Physical state - white crystalline solid
4. Density - 1.5 g/ml
5. Vapor pressure - insignificant

2/ Data obtained from The Ansul Company, Biological Research Center, Weslaco, Texas.

6. Solubility - in H_2O at 20° C. = 25.6% or 25 g/100 ml

7. Stability - stable

8. Melting point - 132-139° C. (pure hexahydrate)

III. EFFICACY DATA UNDER FIELD AND LABORATORY CONDITIONS

A. Effectiveness for Intended Purpose. Newton (1968) reported that results with injections of MSMA were excellent against Douglas-fir, western hemlock, and ponderosa pine; good against bigleaf maple, grand fir, lodgepole pine, and Sitka spruce; and, when mixed with cacodylic acid, excellent against Douglas-fir and good against lodgepole pine and ponderosa pine. Newton and Holt (1968) reported that MSMA is quite efficient against bigleaf maple, but does not greatly affect Oregon white oak. They also stated that MSMA treatments in all seasons provide 80 percent or better control of Douglas-fir and ponderosa pine, although insect activity is least with fall and early winter treatments for the latter species. Spring and fall treatments provide the best control of bigleaf maple. Newton and Webb (1970) stated that MSMA and cacodylic acid are effective in killing young ponderosa pines any season of the year, and that MSMA is cheaper and more effective than cacodylic acid. Lower scolytid attack levels occurred in trees treated with MSMA, cacodylic acid, and a mixture of MSMA and cacodylic acid than in untreated, felled trees (Newton and Hold 1971). Flatheaded borers were common in trees treated with MSMA. Little hatching of Dendroctonus ponderosae occurred in trees treated with MSMA. Flatheaded borer larvae and ambrosia beetles survived all treatments.

Newton and Smith (1971) summarized herbicide injection tests in Vermont from 1966-71. Beech, red maple, and hard maple were injected with Silvisar 510 (cacodylic acid) and Silvisar 550 (MSMA) during August of these years. "All species were readily killed by both Silvisar formulations ....." Red maple and hard maple were quite sensitive to Silvisar 550, while beech was the most resistant species. Some recovery of tree health occurred with Silvisar 510, while damage continued in trees treated with Silvisar 550. Newton and Smith (1971) also stated that recent studies in Tennessee indicate that spaced injections with a Hypo-Hatchet of Silvisar 510 and Silvisar 550 were effective on hardwoods in the fall, but results for the same treatments in winter and spring were less impressive.

B. Persistence in Soil, Water, or Plants. See Section IVB.

C. Compatibility with Other Chemicals. MSMA is compatible with cacodylic acid and 2,4-D.

IV. ENVIRONMENTAL IMPACT

A. Effects on Non-Target Organisms. Evans and Allard (see Norris 1971) determined that the LD_{50} for a commercial formulation of MSMA to snowshoe hares is 173 mg/kg.

Norris (1971) related an incident of snowshoe hare mortality in connection with the use of MSMA as a silvicide. Seven dead animals were found around areas which were used for cleaning application equipment. High arsenic residues were found in soil and vegetation samples from the area. Cardless handling of MSMA may present a hazard to both the applicator and animals.

Morton et al. (1972) fed herbicides to the honeybee, Apis mellifera, in 60 percent sucrose syrup at concentrations of 0, 10, 100, and 1000 parts per million by weight. MSMA was extremely toxic at 100 and 1000 ppmw.

B. Residues in or on Food or Feed or Entering into Food Chain via Air, Water, Soil, Plants, or Animals. Ehman (1965) reported on the effect of high levels of DSMA (disodium methanearsonate) applications to soil on cotton, soybeans, sorghum, and peanuts. The DSMA was applied at rates of 9.5, 31.5, and 63 lb/acre (equivalent to 2, 7, and 14 years of use in cotton, two applications of 2.25 lb/acre/year). When cotton, soybeans, sorghum, and peanuts were planted on the day of treatment, only the peanuts had to be replanted. The second planting of peanuts and the original planting of cotton, soybeans, and sorghum all developed normally. There was some slight stunting at the 63 lb/acre level in the early stages of growth. All high samples showed arsenic residue from 0.29 to 3.64 ppm for treated samples (controls 0.10 to 0.18 ppm). Peanuts and sorghum grain contained low residues at the 9.5 lb/acre rate. At the high rates, residues varied from 0.52 to 3.12 ppm. Cotton seed contained residues at the 31.5 and 63 lb/acre rates. There was no arsenic residues in the soybeans from any of the plots.

Ehman (1965) found that when a combination of 10 lb/acre of cacodylic acid and 10 lb. of DSMA were used, in grapefruit orchards, no residues could be found in the fruit. In soil build-up tests, utilizing 15, 22, 41, and 79 lb/acre of DSMA, no arsenic residues were found in grapefruit.

A few studies have been conducted on organic arsenic residues in grasses (Long et al. 1962; Lucas 1964). A wide range in arsenic residue on coastal Bermuda grass has been found (Searcy and Patterson 1964; McBee et al. 1967). When calcium acid methanearsonate (CAM)

was applied at the rate of 5 lb. of arsenic per acre, the arsenic content went from 114 ppm at 5 days to 5 ppm at 33 days. In comparison, monosodium acid methanearsonate (MSMA), at the same application level, fell from 1921 ppm at 7 days to 38.9 ppm at 36 days. Disodium methanearsonate (DSMA) (4 lb. of arsenic per acre) was more persistent. The amount of arsenic fell from 475.2 ppm at 5 days to 101.8 at 33 days.

Johnson and Hiltbold (1969) found concentrations of As in several crop plants ranging from 1.6 ppm to 5.2 ppm in soils receiving MSMA, DSMA, or MAMA at rates ranging to 8 pounds per acre. DSMA was absorbed by foliage of Bermuda grass and translocated towards leaf tips and roots. Uptake from soil was much less. Arsenic residues declined from 100 ppm to 35 ppm in 30 days in Bermuda grass treated with 2 lb/acre DSMA. Arsenic residues in roots increased to 80 ppm in the same period (Duble et al. 1969).

Newton (see Norris 1971) treated conifers with organic arsenicals in a thinning study in November. Foliage samples contained 110 ppm, 139 ppm, and 58 ppm the following April, June, and August, respectively. Allard (see Norris 1971) measured 116 ppm As in dead pine needles and 2.5 ppm As in green needles from a treated tree. These data indicate needle fall from treated trees is a significant source of arsenic which will enter the forest floor. Norris (personal communication) finds MSMA and cacodylic acid are leached fairly quickly through 3-inch columns of chopped ponderosa pine, Douglas-fir, or mixed true fir-larch needles. In soil, he finds MSMA is quite resistant to leaching. Cacodylic acid is more mobile, but not to the extent that contamination of ground water is a problem. Canutt and Norris (see Norris 1971) have not found detectable quantities of As in streams flowing from areas thinned with MSMA.

Von Endt et al. (1968) incubated C^{14} labeled MSMA in four soil types and found 1.7 to 10 percent degradation of the MSMA in 60 days. The probable degradation product of this reaction is an inorganic arsenate which is inherently more toxic than MSMA; however, inorganic arsenic compounds are much less available for uptake by plants or soil microorganisms and may, in fact, represent less hazard than the MSMA.

Newton (1971) has reviewed the metabolism of the organic arsenicals and suggests that arsine or alkyl arsine are logical products of the microbial metabolism of MSMA and cacodylic acid. While the arsines are fairly toxic, they are also gases and would be expected to leave treatment areas in low concentrations in mass air movement. The production of arsine analogs under field conditions has not been demonstrated.

A number of studies have examined the soil behavior of MSMA and cacodylic acid. Dickens and Hiltbold (1967) showed DSMA was extensively adsorbed by various soils from water solutions of the herbicide. Soils with higher clay content adsorbed more DSMA. No DSMA leached through a 10-inch column of clay soil with 20 inches of water, while 52 percent of applied DSMA leached through a 10-inch column of loam. The remainder of the herbicide appeared to be tightly bound to the soil. Dickens and Hiltbold (1967) also demonstrated up to 16 percent dimethylation of DSMA in soil in 30 days. Woolson et al. (1969) reports organic and inorganic arsenic behavior similarly in soil. They find soils high in aluminim and iron bind arsenic tightly and reduce its availability, in a sense, detoxifying the arsenic. They show, for instance, the water soluble (available) arsenic level in a clay loam is decreased by 90+ percent in 4 weeks after application.

Ehman (1965) found that when an amount of disodium methanearsonate (DSMA) equivalent to 28 lb/acre was applied to the top of a soil column which was leached with 60 inches of water, less than 10 percent of the applied DSMA showed up in the leachate. When sandy loam was used in the soil column, the figure was less than 6 percent. In a similar experiment performed with 15 lb/acre of cacodylic acid, and using an extrapolation to 60 inches of leaching water, about 9 percent leached through the sand column and 6 percent for the sandy loam. It is evident that DSMA and cacodylic acid are largely inactivated by the soil.

LITERATURE CITED

Dickens, R., and A. E. Hiltbold. 1967. Movement and persistence of methanearsonates in soil. Weeds 15: 299-304.

Duble, R. L., E. C. Hold, and G. G. McBee. 1969. Translocation and breakdown of DSMA in coastal bermudagrass. J. Agr. Food Chem. 17: 1247-1250.

Ehman, P. J. 1965. The effect of arsenical buildup in the soil on subsequent growth and residue content of crops. Southern Weed Contr. Conf. Proc. 18.

Frost, D. V. 1970. Tolerances for arsenic and selenium: A psychodynamic problem. World Rev. of Pest Contr. Spring 1970, 9(1): 6-27.

Johnson, L. R., and A. E. Hiltbold. 1969. Arsenic content of soil and crops following use of methanearsonate herbicides. Soil Sci. Soc.Amer. Proc. 33: 279-282.

King, H., and R. J. Ludford. 1960. The relation between constitution of arsenicals and thin action on cell division. J. Chem. Soc. 2086-2088.

Long, J. A., W. W. Allen, and E. C. Holt. 1962. Control of nutsedge in bermudagrass turf. Weeds 10: 285-287.

Lucas, R. E. 1964. Ansar 184 disodium methyl-arsonate and related compounds as selective johnsongrass herbicides in cotton. Southern Weed Contr. Conf. 17: 62-66.

McBee, G. G., P. R. Johnson, and E. C. Holt. 1967. Arsenic residue studies on coastal bermudagrass. Weeds 15: 77-79.

Meliere, K. A. 1959. Cacodylic acid. U.S. Army Engineering Command, Army Chemical Center. ECNR No. 34, June 1959, AD 318626.

Morton, H. L., J. O. Moffett, and R. H. MacDonald. 1972. Toxicity of herbicides to newly emerged honey bees. Environ. Entomol. 1(1): 102-104.

Mrak, E. M. 1969. Report of the Secretary's Commission on pesticides and their relationship to environmental health. U.S. Dept. HEW.

Newton, M. 1968. Chemical Silviculture. Symposium: Management of young growth Douglas-fir and western hemlock. Oregon State University, Corvallis, Ore. 21-29.

Newton, M. 1971. Organic arsenicals: Breakdown in forest trees and in media containing energy sources--a progress report to Environmental Protection Agency, Aug. 26, 1971. Oregon State University, Corvallis, Ore.

Newton, M., and H. A. Holt. 1968. Hatchet-injection of phenoxys, picloram and arsenicals for control of some hardwoods and conifers. Proc. of the Western Soc. of Weed Sci. 22:20-21.

Newton, M., and H. A. Holt. 1971. Scolytid and buprestid mortality in ponderosa pines injected with organic arsenicals. J. Econ. Entomol. 64(4): 952-8.

Newton, M., and R. W. Smith. 1971. Chemical silviculture in northern hardwoods: A summary of herbicide treatments on the Shatterack Forests Property involving injection and aerial spraying. Office Report, Oregon State University, Corvallis, Ore. 5 p.

Newton, M., and W. L. Webb. 1970. Herbicides and management of young pine. From symposium on regeneration of ponderosa pine held at Oregon State University, Corvallis, Ore, Sep. 11-12, 1969. 94-99.

Norris, L. A. 1971. Studies of the safety of organic arsenical herbicides as precommercial thinning agents: A progress report. Precommercial thinning in the Pacific Northwest. Washington State University, Pullman, Wash. p. 63-74.

Pinto, S. S., and B. M. Bennett. 1963. Effect of arsenic trioxide exposure on mortality. Arch. of Environ. Health 7: 583-591.

Rostand, J. 1950. Chemical teratogenesis in anurian batrachians. Compt. Rend. Soc. Biol. 144: 915-917.

Salzgeber, B. 1955. Modification observed in chick embryo genital organs inplanted in vitro after treatment with different teratogenic substances. Compt. Rend. Soc. Biol. 149: 190-192.

Searcy, V. S., and R. M. Patterson. 1964. Weed control in establishment of coastal bermudagrass. Southern Weed Contr. Conf. Proc. 17: 106.

USDA. 1967. Agr. Handbook No. 332. ARS.

Von Endt, D. W., P. C. Kearney, and D. D. Kaufman. 1968. Degradation of MSMA by soil microorganisms. J. Agr. Food Chem. 16: 17-20.

Woolson, E. A., P. C. Kearney, and J. H. Axley. 1969. Chemical distribution of arsenic in soil. Am. Chem. Soc. Mtg., N.Y., N.Y.

REPORT

ON

BACKGROUND INFORMATION

FOR

PICLORAM

PICLORAM - BACKGROUND INFORMATION STATEMENT

Prepared by Robert F. Buttery, Timothy R. Plumb and Kenneth D. Weyers

I. General Information

A. Common name(s) - Picloram, Tordon, ATCP

B. Chemical name - 4-amino-3,5,6-trichloropicolinic acid

C. Registered uses - Control of annual and deep rooted perennial weeds in noncropland.

D. Formulation(s) manufactured -

1. Tordon 101 mixture

Active Ingredients:

4-amino-3,5,6-trichloropicolinic acid as the triisopropanolamine salt	10.2%
2,4-dichlorophenoxyacetic acid as the triisopropanolamine salt	39.6%
Inert Ingredients	50.2

Acid Equivalents:

4-amino-3,5,6,-trichloropicolinic acid	5.7%
2,4-dichlorophenoxyacetic acid	21.2%

U.S.D.A. Registration No. - 464-306

2. Tordon 10K Pellets

Active Ingredients:

4-amino-3,5,6-trichloropicolinic acid as the potassium salt	11.6%
Inert Ingredients:	88.4%

Acid Equivalent:

4-amino-3,5,6-trichloropicolinic acid	10%

U.S.D.A. Registration No. - 464-320

3. Tordon 22K Weed Killer

Active Ingredient:

4-amino-3,5,6-trichloropicolinic acid as the potassium salt	24.9%
Inert Ingredients	75.1%

Acid Equivalents:

4-amino-3,5,6-trichloropicolinic acid (2 lbs./gal.)	21.5%

U.S.D.A. Registration No. - 464-323

4. Tordon Beads

Active Ingredients:

4-amino-3,5,6-trichloropicolinic acid as the potassium salt	2.3%
Disodium tetraborate pentahydrate	79.2%
Disodium tetraborate decahydrate	16.5%
Inert Ingredients:	2.0%

Acid Equivalents:

4-amino-3,5,6-trichloropicolinic acid	2.0%
Boron Trioxide	43.8%

U.S.D.A. Registration No. - 464-333

5. Tordon 212 Mixture
Active Ingredients:
4 - amino - 3,5,6 - trichloropicolinic acid as the triisopropanolamine salt - - - - - - - - - - - - - - 18.1%
2,4-dichlorophenoxyacetic acid as the triisopropanolamine salt - - - - - - - - - - - - - - - - 37.7%
Inert Ingredients - 44.2%
Acid Equivalents:
4 - amino - 3,5,6 - trichloropicolinic acid - - - 10.1%-1 lb./gal.
2,4 dichlorophenoxyacetic acid - - - - - - - - - - 20.2%-2 lb./gal.
U.S.D.A. Registration No. - - 464-361

6. Tordon 155 Mixture
Active Ingredients:
4 - amino - 3,5,6 - trichloropicolinic acid as the isooctyl ester - 15.1%
2,4,5-trichlorophenoxyacetic acid as the propylene glycol butyl ether esters - - - - - - - - - - 63.4%
Inert Ingredients 21.5%
Acid Equivalents:
4 - amino - 3,5,6 - trichloropicolinic acid - - - 10.3%-1 lb./gal.
2,4,5 - trichlophenoxyacetic acid - - - - - - - - 41.3%-4 lbs./gal.
U.S.D.A. Registration No. 464-364

E. Dilution of formulations and rate and method of application.
1. Tordon 101 Mixture:
Use Tordon 101 Mixture at rates of 1/2 to 3 gallons per acre to control broadleaved weeds and at rates of 1 to 4 gallons per acre to control woody plants and vines. In all cases use the amounts specified in enough water to give thorough and uniform coverage of the plants to be controlled. NOTE: Tordon 101 Mixture does not mix readily with oil.

For best results applications should be made when weeds and brush are actively growing. Applications in late summer when the plants are mature or during period of drought may result in less effective control. Treatment will not cause permanent, if any, damage to common established grasses.

High Volume Leaf-Steam Treatment: Use Tordon 101 Mixture at the rate of 1 gallon in water to make 100 gallons of spray to control broadleaved weeds, vines and other woody plants. Apply after the foliage is well developed and in a manner to give thorough spray coverage. For woody plants, up to 6 to 8 feet tall, use a drenching spray and wet all leaves, stems, and root collars. For hard to kill species such as ash and oak soak the soil around the root collar. NOTE: Do not allow the spray to contact desirable plants, and do not soak the soil over roots of such plants.

Low Volume Ground or Aerial Foliage Treatment: For these uses the required amount of Tordon 101 Mixture should be applied in a total spray volume of 10 to 25 gallons per acre, depending upon the plant species, height and density of growth. The preferred volume range is 15 to 25 gallons per acre. For these Low Volume uses, Tordon 101 Mixture should be used only in thickened (high viscosity) spray mixtures. Such mixtures should be prepared using NORBAK particulating agent as directed in a separate publication "INSTRUCTION MANUAL FOR NORBAK PARTICULATING AGENT WITH HERBICIDES" (available from the Dow Chemical Company) and in the accompanying "GUIDE TO INGREDIENT NEEDS AND PROCEDURES TO FOLLOW FOR MIXING SPRAYS CONTAINING TORDON 101 MIXTURE PLUS NORBAK PARTICULATING AGENT." Thickened sprays prepared by using high viscosity invert emulsions or other drift reducing systems may be utilized if they are made as drift-free as are mixtures containing NORBAK particulating agent mixed according to manufacturer's directions.

Broadleaved Annual and Perennial Weed and Woody Vine Control: Use Tordon 101 Mixture at rates of 2 quarts to 3 gallons per acre in 15 to 25 gallons of a water spray mixture containing the amount of NORBAK particulating agent required to provide the recommended thickness. Apply to problem weeds and vines any time after growth begins in the spring and before the ground freezes in the fall. For seasonal control of vigorously growing stands of field bindweed, Canada thistle or mixtures of these with susceptible annual weeds such as ragweed, dandelion, plantain, clovers and dock use 2 to 3 quarts of Tordon 101 Mixture per acre in 15 to 25 gallons of water spray containing NORBAK particulating agent. In arid areas and for control of more resistant perennial weeds use 1 to 3 gallons of Tordon 101 Mixture per acre in 15 to 25 gallons of spray containing NORBAK particulating agent. Use 1 to 1.5 gallons per acre to control species such as Canada thistle, field bindweed and milkweed. The higher rates should be used under drought stress conditions and for the more resistant species such as bouncingbet, leafy spurge, toadflax and woody vines.

Woody Plant Control: Use Tordon 101 Mixture at the rate of 1 to 4 gallons per acre in 15 to 25 gallons of a water spray mixture containing NORBAK particulating agent. For susceptible seedling stages of species such as aspen, cherry and sumac use 1 to 1.5 gallons of Tordon 101 Mixture per acre in 15 to 25 gallons of a water spray mixture containing NORBAK particulating agent. For more mature and/or less susceptible species such as willow, buttonbush, black locust, sassafras, sumac, tulip poplar and cherry growing in sandy loam soil, use 2 to 2.5 gallons of Tordon 101 Mixture per acre in 15 to 25 gallons of a water spray mixture containing NORBAK particulating agent.

For more resistant brush such as maple, pine, sourwood, blackgum, cedar and oak where growing on heavy clay soils or on rocky terrain, use 3 to 4 gallons of Tordon 101 Mixture per acre in 15 to 25 gallons of a water spray mixture containing NORBAK particulating agent. Use the higher rate and volume where the foliage of more difficult to kill brush is covered with dense vine growth. NOTE: For best results under conditions of drought stress use the higher rates recommended. Even these rates under such conditions may not be as effective as the lower rates under good growing conditions.

Cut Surface Treatments: In forest and other non-crop areas to kill unwanted trees of hardwood species such as elm, maple, oak and conifers such as pine apply Tordon 101 Mixture, either undiluted or diluted in a 1 to 1 ratio with water, as directed below.

With Tree Injector Method: Application should be made by injecting 1/2 milliliter of undiluted Tordon 101 Mixture or 1 milliliter of the diluted solution through the bark at intervals of 3 inches between edges of the injector wound. The injections should completely surround the tree at any convenient height.

With Frill or Girdle Method: Make a single girdle through the bark completely around the tree at a convenient height. Wet the cut surface with the diluted solution.

Both above methods may be used successfully at any season except during periods of heavy sap flow of certain species - for example maples.

2. Tordon 10K Pellets:

Tordon 10K Pellets may be applied at any time soil is free from frost. However, best results are obtained from applications in the spring before growth begins or during periods of vigorous growth and when abundant rainfall can be expected. Distribute Tordon 10K Pellets uniformly as spot or broadcast treatment to the soil over the roots of woody plants to be controlled.

Broadcast application is the preferred method of treatment for dense stands of brush. Use Tordon 10K Pellets at the rate of 60 to 85 pounds per acre (approximately 1-1/2 to 2 lbs. per 1000 sq. ft.) and distribute evenly over the entire area where brush is to be controlled.

To control solid stands of very susceptible species such as maple, conifers, locust, aspen and wild rose use Tordon 10K Pellets at the rate of 60 lbs. per acre.

To control brush of mixed species use Tordon 10K Pellets at the rate of 75 lbs. per acre.

To control solid stands of hard to kill species such as blackgum, oak and ash use Tordon 10K Pellets at the rate of 85 lbs. per acre. Re-treatment of ash may be necessary the following year.

Spot application is generally the preferred method of treatment for scattered or sparse stands of brush. Spread Tordon 10K Pellets evenly on the soil over the entire root system (around the main stem) and outward to 1 foot beyond the branch tips (drip-line). Use at the rate of 1 to 2 tablespoonfuls (1 to 2 ounces) per 30 square feet of soil surface.

Use the higher dosage also to control brush on very sandy, gravelly or rocky soils and in areas where heavy rainfall can be anticipated.

3. Tordon 22K Weed Killer:

Mix with water and apply as a coarse, low pressure spray (20 to 40 lbs. per sq. in.). Apply anytime during the growing season (when frost leaves soil in spring until ground freezes in fall), and preferably when rainfall can be expected soon after application.

For General Use on Perennial Weeds on Non-cropland, use 1 to 1-1/2 gallons of Tordon 22K Weed Killer per acre in 50 to 100 gallons of water and spray to wet weed foliage and soil. NOTE: Local conditions may affect the use of herbicides. State agricultural experiment stations or extension service weed specialists in many states issue recommendations to fit local conditions. Be sure that use of this product conforms to all applicable regulations.

For Use as a Spot Treatment on Perennial Weeds. Mix at the rate of 1 gallon of Tordon 22K per 100 gallons of water. Apply at the rate of 100 gallons of spray mixture per acre. This will provide a rate of 2 pounds of Tordon herbicide per acre. For small amounts us 2-1/2 fluid ounces Tordon 22K per 2 gallons of water. For round patches apply as indicated in the table.

Feet across Round Patch to be treated (weed area plus 10 foot border)	Gallons of spray mixture to apply
25	1.0
50	4.5
75	10.0
100	18.0
235 or (1 acre)	100.0

4. Tordon Beads:

Tordon Beads herbicide applied to the soil over plant roots is highly effective for the control of broadleaved perennial and annual weeds and undesirable woody plants on utility, highway and other right-of-ways, fencerows, headlands around farm and industrial buildings and storage sites.

For Control of Broadleaved Perennial and Annual Weeds: Apply Tordon Beads uniformly anytime during the normal growing season where sufficient moisture is available to carry the herbicide into the soil. In areas where little or no summer rainfall occurs, application should be made in late summer or early fall. Maximum effects of the treatment do not become apparent until the chemical has been carried by moisture into the soil.

APPLICATION RATES

Weeds Controlled*	Tordon Beads Amount to Apply	Remarks
Docks Larkspur Pigweed Povertyweed Sowthistle (perennial) Sunflower Tansy Thistle (plumeless) Toadflax (dalmation)	50 to 100 lb. per acre 19 to 37 oz. per 1000 sq. ft. 5 to 10 oz. per sq. rod	Use lower rates in low rainfall areas in the northern states such as Idaho, Montana, North Dakota, Oregon, South Dakota, Wyoming and Washington. Higher rates should be used where rainfall is greater or in southern states such as Arizona, Arkansas, Kansas Missouri, New Mexico, Oklahoma and Texas.
Bindweed (field) Bursage (bur-ragweed woolyleaf povertyweed) Knapweed (Russian) Milkweed Spurge (leafy) Thistle (Canada)	100 to 150 lb. per acre 37 to 56 oz. per 1000 sq. ft. 10 to 16 oz per sq. rod	

*These are types or examples of weeds controlled

Tordon Beads herbicide is effective against a wide range of weeds. Local conditions may affect the use of herbicides. Consult your State Agricultural Experiment Station or Extension Service weed specialists for local recommendations. Be sure that the use of this product conforms to all applicable regulations.

For Control of Woody Plants such as maple, locust, aspen, conifers, other woody trees, shrubs, wild rose, brambles, wild grapes and other vines, apply Tordon Beads uniformly to the soil over the root zone. Apply anytime during the normal growing season where sufficient moisture is available to carry the herbicide into the soil in areas where little or no summer rainfall occurs application should be made at "bud break" in late winter or early spring. Use at the rate of 300 to 400 pounds per acre (equivalent to approximately 7-1/2 to 10 lb. per 1000 square feet, 2 to 2-1/2 lb. per sq. rod, or 1/4 to 1 lb per 100 sq. ft.) Maximum effects of the treatment do not become apparent until the chemical has been carried by moisture into the soil in the root zone of the plants.

5. Tordon 212 Mixture

Mix with water and apply as a coarse, low pressure spray (20 to 50 lbs. per sq. in.). Apply anytime when fully developed green leaves are present.

For General Use: The rate of Tordon 212 Mixture required varies according to weed species and geographical location. The following table shows the amount of Tordon 212 Mixture that should be mixed in water to make 100 gallons of spray. Apply uniformly to wet the weeds without run-off. This will usually require about 100 gallons per acre.

Some of the Weeds to be Controlled	Tordon 212 Mixture to use in 100 gal. spray	Remarks
Dock, Larkspur Pigweed Sowthistle Sunflower (wild) Thistle (Canada) Thistle (Musk) Toadflax (Dalmation) Wormwood (American)	1/2 to 2 gallons	Use lower rates in low rainfall areas in the northern states such as Idaho, Montana, North Dakota, Oregon, South Dakota, Washington and Wyoming. Higher rates should be used in southern states or where rainfall is greater such as Arizona, Arkansas, Kansas, Missouri, New Mexico, Oklahoma, and Texas.
Bindweed (field) Horsenettle (white) Knapweed (Russian) Milkweed Ragweed (bur) Spurge (leafy) Toadflax (yellow)	1 to 3 gal.	

For Use on Round Patches of Weeds: Apply the required spray mixture at the amount indicated in the following table.

Feet across Round Patch to be treated (weed area plus 10 foot border)	Gallons of spray mixture to apply
25	1.0
50	4.5
75	10.0
100	18.0
235 (or 1 acre)	100.0

NOTE: For small amounts of spray use Tordon 212 Mixture at rate of 1-1/4 to 2-1/2 fluid ounces in each gallon of water.

6. Tordon 155 Mixture:

Basal Bark Treatment: Use 1 to 3 gallons of Tordon 155 Mixture in enough diesel oil, No. 1 or No. 2 fuel oil or kerosene to make 100 gallons of spray mixture. Apply with knapsack sprayer or power spraying equipment using low pressures (20-40 psi). Spray the basal parts of brush and tree trunks to a height of 12 to 15 inches from the ground. Thorough wetting of the indicated area is necessary for good control. Spray until run-off at the ground line is noticeable. Old or rough bark requires more spray than smooth young bark. Apply at any time, including the winter months, except when snow or water prevent spraying to the ground line.

DORMANT STEM BROADCAST: Mix 3 to 6 quarts of Tordon 155 Mixture brush killer in enough oil to make 100 gallons of spray. Apply with knapsack or power spraying equipment, using low pressure (20-40 psi). Treat any time when brush is dormant and most of the foliage has dropped. Thoroughly wet the upper parts of the stems and use the remainder needed to wet the lower 12 to 15 inches above the ground to the point of run-off. For root suckering species such as sumac, persimmon, sassafras and locust, also spray the ground under the plants to cover small root suckers which may not be visible above the soil surface. Brush of average density and 4 to 6 feet high may take up to 150 gallons of spray mixture per acre.

F. Tolerances in food or feed and other safety limitations.

Food or Feed Item	Tolerance (parts/million)
Forage grass	80
Kidney	5
Liver	0.5
Meat fat and byproducts	0.2
Milk	0.5

Safety limitations for the different Picloram are as follows:

1. Tordon 101 Mixture:

Do Not Allow Spray Drift: Tordon 101 Mixture is highly active against most broadleaved plants. Tiny amounts may cause injury to such plants if applied during either growing or dormant periods. Do not use high pressure sprays. Do not apply or otherwise permit Tordon 101 Mixture or sprays containing it to contact desirable plants such as flowers, other ornamental plants, vegetables, grapes, fruit trees, cotton, tobacco, tomatoes, potatoes, beans of all types including soybeans, and other valuable broadleaved plants, nor the soil containing roots of such valuable plants. Apply Tordon 101 Mixture only when there is little or no wind and no hazard from drift. Coarse sprays are least likely to drift.

Do Not Contaminate Water: To avoid injury to crops or other desirable plants, do not treat or allow spray drift to fall onto inner banks or bottom of irrigation ditches.

Other Precautions: Do not store near food, feedstuff, fertilizer, seeds, insecticides, fungicides or other pesticides. To avoid injury to desirable plants, containers and sprayers used for Tordon 101 Mixture should not be reused to contain or apply other materials.

Rinse equipment and containers thoroughly with water and dispose of wastes by burying in non-croplands away from water supplies. Containers should be disposed of by punching holes in them and burying with waste.

CAUTION: KEEP OUT OF REACH OF CHILDREN. HARMFUL IF SWALLOWED. CAUSES EYE INJURY. MAY CAUSE SKIN IRRITATION. Avoid Contact with Eyes, Skin and Clothing, Wash Well After Handling or Use. Keep Container Closed. Keep Away from Heat and Open Flame.

When handling concentrate wear suitable eye protection. In case of eye contact, promptly flush with plenty of water, and get medical attention. Remove contaminated clothing and wash before reuse. COMBUSTIBLE LIQUID.

2. Tordon 10K Pellets:

Apply only as recommended to avoid injury to desirable plants. Avoid application during windy periods when the product may be blown from area to be treated.

Do not clean containers or application equipment over or near areas where roots of desirable trees and other desirable plants may extend into the soil where the chemical may be washed or otherwise moved into contact with the roots.

Do not permit any of the product to be blown onto any parts of desirable plants.

Do not allow the material to contaminate water used for irrigation, drinking or other domestic purposes.

Do not store near food, feedstuff, fertilizers, seeds, insecticides, fungicides or other pesticides.

Equipment used for applying Tordon 10K Pellets should not be used for applying other materials to desirable plants. Shipping containers should not be re-used for other materials which may be applied to desirable plants. Dispose of empty containers by burning or burying in non-croplands away from desirable plants and water supplies.

NOTE: Be sure that all use of Tordon 10K Pellets conforms to local regulations.

CATUION: Keep out of Reach of Children.

3. Tordon 22K Weed Killer

Do Not Allow Spray Drift. Tordon herbicide is highly potent. Tiny amounts may cause damage to plants if applied during either growing or dormant periods. Do not use high pressure sprays. Do not apply by aerial equipment. Do not apply or otherwise permit Tordon 22K or sprays containing it to contact desirable plants such as vegetables, flowers, grapes, fruit trees, ornamentals, cotton, tobacco, tomatoes, potatoes, beans of all types including soybeans, and other valuable broadleaved plants, nor the soil containing roots of nearby valuable plants. Apply Tordon 22K only when there is little or no wind or no hazard from spray drift. Coarse sprays are least likely to drift.

Do Not Contaminate Water. To avoid crop or other plant injury, do not treat or allow spray drift to fall onto inner banks or bottom of irrigation and drainage ditches. Dike around and do

not irrigate through treated areas. Do not contaminate water used for drinking or other domestic purposes.

Do Not Move Treated Soil. Do not go over treated areas with land levelers, cultivation or harvesting equipment, or move the soil by any other means. Mark off treated areas with stakes, posts or fencing.

Do Not Graze Or Use Treated Areas for Crop Production.

Do Not Mix With Other Weedkillers or Other Pesticides.

Other Precautions: Do not store near food, feedstuff, fertilizers, seeds, insecticides, fungicides or other pesticides. To avoid injury to desirable plants, containers and sprayers used for Tordon 22K should not be reused to contain or apply other materials. Be sure that all use of Tordon 22K conforms to local regulations.

CAUTION - MAY CAUSE IRRITATION - COMBUSTIBLE. Avoid Contact with Skin and Eyes. Avoid Breathing Spray Mist. Keep Container Closed. Keep Away from Heat and Open Flame. Keep Out of the Reach of Children.

4. Tordon Beads:

USE PRECAUTIONS:

Avoid Improper Application: Tordon herbicide is highly active against most broadleaved plants. Small quantities may cause damage to plants whether applied during the growing or dormant season. Do not apply or otherwise permit Tordon Beads to contact desirable plants such as vegetables, flowers, grapes, fruit trees, ornamentals, cotton, beans, soybeans and other valuable broadleaved plants nor the soil containing roots of such plants growing thereon or nearby or where such plants are to be grown.

Avoid Water Contamination: To avoid crop or other plant injury, do not treat inner banks or bottom of irrigation and drainage ditches. Do not contaminate water to be used for drinking or other domestic purposes.

Avoid Movement of Treated Soil: Avoid the movement of treated soil into untreated areas.

Other Precautions: Do not store near food, feedstuffs, fertilizer, seeds, insecticides, fungicides or other pesticides. To avoid injury to desirable plants, containers and equipment used for Tordon Beads should not be re-used to contain or apply other materials.

Dispose of empty containers: Burn or bury in non-cropland away from desirable plants and water supplies.

CAUTION: DUST CAUSES IRRITATION. MAY BE HARMFUL IF SWALLOWED. KEEP OUT OF REACH OF CHILDREN. Avoid Skin and Eye Contact. Wash After Handling.

5. Tordon 212 Mixture:

Do Not Allow Spray Drift. Tordon and 2,4-D herbicides are highly potent. Tiny amounts may cause damage to plants if applied during either growing or dormant periods. Do not use high pressure sprays. Do not apply or otherwise permit Tordon 212 Mixture or sprays containing it to contact desirable plants such as vegetables, flowers, grapes, fruit trees, ornamentals, cotton, tobacco, tomatoes, potatoes, beans of all types including soybeans, and other valuable broadleaved plants, nor soils containing roots of nearby valuable plants. Apply Tordon 212 Mixture only when there is little or no hazard from spray drift. Coarse sprays are least likely to drift. Do not apply by air, as this increases drift hazard.

Do Not Contaminate Water. To avoid crop or other plant injury, do not treat or allow spray drift to fall onto inner banks or bottom of irrigation and drainage ditches. Dike around and do not irrigate through treated areas. Do not contaminate water used for drinking or other domestic purposes.

Do Not Move Treated Soil. Do not go over treated areas with land levelers, cultivation or harvesting equipment or move soil from treated areas by any other means.

Do Not Treat Areas intended to be used for desirable plants or Food Crops, It usually requires up to 3 years for Tordon herbicide to be deactivated by the soil.

Do Not Mix in the Sprayer with Other Weedkillers or Other Pesticides.

Other Precautions: Do not store near food, feedstuff, fertilizer, seeds, insecticides, fungicides or other pesticides. To avoid injury to desirable plants, containers and sprayers used for Tordon 212 Mixture should not be reused to contain or apply other materials. Rinse equipment and containers thoroughly with water and dispose of wastes by burying in non-croplands away from water supplies. Containers should be disposed of by punching holes in them and burying with waste.

CAUTION. KEEP OUT OF REACH OF CHILDREN. HARMFUL IF SWALLOWED. CAUSES EYE INJURY. MAY CAUSE SKIN IRRITATION. Avoid Contact with Eyes, Skin and Clothing. Avoid Breathing Spray Mists. Wash Well After Handling or Use. Keep Container Closed When Not Using. In case of contact, flush eyes with plenty of water; and get medical attention. Remove grossly contaminated clothing and wash before reuse.

6. Tordon 155 Mixture:

Do Not Use Tordon 155 Mixture With Water. Tordon and 2,4,5-T herbicides are highly potent and even minute quantities may damage plants during both the growing and dormant periods. Therefore, do not apply or otherwise permit Tordon 155 Mixture or spray mist containing it to contaminate soil used to grow desirable susceptible plants nor to contact susceptible plants such as vegetables, flowers, grapes, fruit trees, ornamentals, cotton, beans of all types including soybeans and other desirable broad-leaved plants. Applications should be made only when there is no hazard from spray drift. Coarse sprays are less likely to drift. Do not allow the material to contaminate water used for irrigation, drinking or other domestic purposes. Do not store near food, feedstuff, fertilizer, seeds, insecticides, fungicides or other pesticides. Because of the difficulty of thoroughly cleaning sprayers such equipment should not be used for applying other materials to desirable plants. Shipping containers should not be re-used for other materials which may be applied to desirable plants.

This product is toxic to fish. Keep out of lakes, streams or ponds.

Rinse equipment and containers thoroughly with water and dispose of wastes by burying in non-cropland away from water supplies. Containers should be disposed of by punching holes in them and burying with waste.

NOTE: Be sure that all use of Tordon 155 Mixture conforms to local regulations.

CAUTION. KEEP OUT OF THE REACH OF CHILDREN. HARMFUL IF SWALLOWED. MAY CAUSE IRRITATION. Avoid Contact with Eyes, Skin and Clothing. In case of contact wash with plenty of water.

G. Manufacturer or producer:

The Dow Chemical Company
Midland, Michigan 48640

II. Toxicity Data on Formulation to Be Used

A. Safety data

Based on numerous tests, the recommended use of picloram containing herbicides should present no safety hazard to humans, livestock, or wildlife (143). McCollester and Leng also report that no alarming pharmacological or toxicological properties were found in investigations in animals, fish, and aquatic organisms. Formulations containing phenoxy derivatives appear to be more toxic than picloram alone.

1. Acute mammalian studies.

a. Oral. The acute oral toxicity of picloram to various animals in terms of LD_{50} (lethal dose to kill 50 percent of the animals) values range from 2,000 mg of picloram/kg of body weight in mice and rabbits to 8,200 mg/kg in rats. The LD_{50} value for cattle and sheep are greater than 750 and 1,000 mg/kg respectively (230). A single dose of up to 500 mg/kg gave no evidence of toxicity in calves, and the LD_{50} value for chicks is approximately 600 mg/kg. Lynn (134) reported that sheep showed no ill effect from the acid form of picloram at rates up to 650 mg/kg and the K-salt formulation (25 percent active ingredient) up to 4,650 mg/kg. However, the Tordon 101 formulation (10.7 percent picloram and 39.6 percent 2,4-D as triisopropanolamine salts) produced toxic effects at 2,530 mg/kg and subsequent death in 3 days. Cattle were more sensitive in showing toxic effects, but not death, at a rate of Tordon 101 of 1,900 mg/kg; no death was reported at a rate greater than 3,000 mg/kg. Bovey and Scifres (36) noted that there are no known reports of human sickness resulting from the handling or application of picloram.

b. Dermal. Skin irritation is minimal, and picloram is not likely to be absorbed through the skin. The LD_{50} value for rabbits is greater than 4,000 mg/kg, the highest value tested (230). In a similar test, Tordon 22K at 2,000 mg/kg caused no observable effect while a similar rate of Tordon 101 caused slight hyperemia and slight necrosis (134).

c. Inhalation. Picloram dusts may be somewhat irritating, but they are not likely to cause illness (230). Inhalation of air for 7 hours bubbled through a solution of Tordon 22K produced no observable adverse effects during or within 2 weeks after exposure (134).

d. Eye and skin irritation. Picloram may cause mild irritation to the eyes which heals readily and no corneal injury is likely (230). Undiluted picloram applied directly to the conjunctival sac of white rabbits produced slight redness and slight corneal cloudiness both of which disappeared in 1 to 2 days (134). The Tordon 101 mixture was slightly more irritating.

2. Subacute studies.

a. Oral. Feeding studies for 90 days in rats showed no adverse effects from dietary levels as high as 0.1 percent (1,000 ppm) of picloram (143). The only effect noted at 0.3 percent picloram in the diet was an increase in liver/body weight

ratios of the females. Only slight to moderate pathological changes were observed in the liver and kidneys on a diet containing 1 percent (1,000 ppm) picloram. No adverse effects were noted in any animals fed a 0.3 percent triisopropanolamine salt picloram diet.

In long-term feedings, albino rats and beagle dogs were fed picloram at a rate of 15 to 150 mg/kg of body weight for 2 years. No observable adverse effects were noted in either species as measured by body weight, food consumption, behavior, mortality, hematological and clinical blood chemistry studies, and urine analyses. Also, no pathological differences were found between the incidence or kind of tumors in control and treated animals. No adverse effects were found in sheep or cattle fed picloram at 73 mg/kg/day for 30 days.

b. Dermal. Continued exposure for 9 days of the skin of rabbits to the undiluted acid form of picloram caused only slight exfoliation and hyperemia (134). Other tests where the skin of rabbits was exposed for several days to various concentrations of picloram showed no severe or prolonged effects. Exposure of the skin of human subjects to a 10 percent solution of picloram caused no skin irritation (the duration of the test was not reported).

c. Inhalation. No information available.

3. Other toxicity studies which may be required.

a. Neurotoxicity. No information available.

b. Teratogenicity. Only one brief reference; see section "C" below.

c. Effects on reproduction. No adverse effects were found in albino rats fed picloram at various levels in the diet up to 3,000 ppm. through three generations (two litters per generation) in terms of fertility, gestation, viability, and lactation by body weight records and by teratological examination of the fetuses (143). Mice fed 0.01 percent picloram in their diet for 4 days before mating and 14 days after mating produced the same number of offspring before and after the test.

d. Synergism. No information available.

e. Potentiation. No information available.

f. Metabolism. McCollester and Leng (143) reported that dogs fed on a diet containing 97 ppm picloram (carboxyl-^{14}C-

labeled) excreted 90 percent of the dose unchanged in the urine within 48 hours after feeding. Picloram apparently did not accumulate in the tissue of the animals and neither was it decarboxylated in vivo. Based on work by other investigators (e.g. Fisher et al. 1965), McCollester and Leng concluded that mammals were found to eliminate 98 percent of the picloram from the bloodstream and kidneys as an unchanged compound in the urine before the liver had an opportunity to act on it. Menzie (145) referring to Redemann et al. (179) noted that picloram remained mainly unchanged in spring wheat grown on treated soil; however, metabolites in low levels were found including 4-amino-6-hydroxy-3,5-dichloropicolinic acid, oxalic acid, lipid conjugates, and 4-amino-3,5,6 trichloropyridine.

g. Avian and fish toxicity. Kenaga (118) reported that all derivatives of picloram exhibit low acute toxicity to birds and fish. If the recommended use directions are followed, there is low potential hazard, if any, to fish from terrestrial runoff water or from direct accidental contamination of water and there is no hazard to birds.

Japanese and Bobwhite quail (Coturnix couturnix japonica and Colinus virginianus respectively) and mallard ducks (Anas platyrhynchos) fed picloram at rates up to 1,000 ppm or more did not receive the LC_{50} (median lethal concentration) values. Bobwhite quail and mallard ducks had a calculated LC_{50} dosage of 23,000 and 385,000 ppm respectively. Japanese quail were fed up to 1,000 ppm of picloram in their diet for each of three successive generations without effect on mortality, egg production, and fertility.

Kenaga (118) also reported on the effect of picloram on 15 species of fish including rainbow trout (Salmo gairdnerii Richardson) and channel catfish (ictalurus melas Rafinesque). Picloram formulations as acids, salts, and esters were generally low in toxicity to fish ($LC_{50} > 1.0$ ppm). Assuming that all material was completely dissolved, a 3 pound application of picloram to an acre of water 3 inches deep would result in a maximum concentration of 4.5 ppm. This is less than the LC_{50} values of the picloram salt formulations to the fish studied. However, the isooctyl ester would be toxic to the most sensitive species which had an LC_{50} value around 1 ppm. Kenaga noted that picloram herbicides are not recommended for aquatic uses. Land applications of 3 pounds a.e. per acre would not likely result in concentrations as high as 1 ppm in water whether by accidental application or by runoff because of the dilution, sorption, and degradation that occurs.

Referring to other work, Kenaga noted that a 90-day exposure of bluegill (Lepomis macrochirus Rafinesque) to 5 to 8 ppm of picloram resulted in a 30 percent kill and some loss of weight in the survivors. Hardy (104) studied the effect of the K-salt of picloram on the biological food chain of algae-daphnia-fish. The presence of 1 ppm of picloram did not retard algae growth. Daphnia which were maintained in 1 ppm a.e. of picloram for 10 weeks developed and reproduced normally with no build-up of herbicide in their tissue. Guppies kept in water at 1 ppm a.e. of picloram and fed a diet of daphnia reared in a similar picloram solution appeared normal in development, behavior, and reproduction.

h. Carcinogenicity. No information available.

B. Physical-chemical properties of the pure chemical (4-amino-3,5,6-trichloropicolinic acid).

1. Boiling point: Decomposes at approximately 215 C.

2. Flash point:
   a. Pure chemical -- ?
   b. Tordon 101 Mixture--35 C TOC
   c. Tordon 10K Pellets--Nonflammable.
   d. Tordon 22K Weed Killer--Combustible (flashpoint unknown).
   e. Tordon Beads--Nonflammable
   f. Tordon 212 Mixture--Nonflammable
   g. Tordon 155 Mixture--140 C COC.

3. Physical state: White powder with a chlorine-like odor.

4. Density: No information.

5. Vapor pressure:
   a. 6.16×10^{-7} MM Hg at 35 C
   b. 1.07×10^{-6} MM Hg at 45 C

6. Solubility: At 25 C

Solvent	g/100 Ml Solvent	ppm
Acetone	1.98	19,800
Acetonitrile	0.16	1,600
Benzene	0.02	200
Carbon disulfide	less than 0.005	Less than 50
Diethyl ether	0.12	1,200
Ethanol (2B absolute)	1.05	10,500
Isopropanol	0.55	5,500
Kerosene	0.001	Less than 10
Methylene chloride	0.06	600
Water	0.043	430

7. Stability: No information.

III. Efficacy data under field and laboratory tests

A. Effectiveness for intended purpose

Picloram alone (Tordon 22K) or mixtures with phenoxy herbicides (Tordon 212 and 225) have been the only effective treatment for the control of Gamble oak (Quercus gambellii Nut.) in southwest Colorado. Rates of Tordon 22K up to 2 pounds have given up to 80 percent stem kill and it is the only single treatment giving comparable results to Tordon 22K plus silvex (Kuron) (139). Gantz and Warren (80) found that picloram plus 2,4-D, at 1/4 plus 4 oz. per acre respectively, gave satisfactory control of wild buckwheat (Polygonum convolvulas) in spring wheat, barley, and winter wheat with adequate crop safety. Picloram at rates of 3 and 4 pounds per acre killed almost all plants of brush species in Texas except Texas persimmon; lower rates were effective on honey mesquite, pricklypear, and whitebrush (74). In California, a single broadcast application of 2 pounds a.e. per acre of picloram gave approximately 85 percent kill of chamise (Adenostoma fasciculatum) (172); while in other work three annual broadcast applications of 6 pounds a.e. per acre of picloram gave less than 50 percent plant kill of scrub oak (Quercus dumosa) (173).

Soil application of Tordon to control woody plants should be made prior to or during early spring growth and when rain is expected afterward. Treatment at other times may be effective, but higher dosages may be required.

Many annual broadleaf weeds can be killed with foliar applications of Tordon at rates as low as 1/4 to 1/2 ounce per acre.

Most established perennial grasses are not affected by rates of 1 to 2 pounds per acre. A large number of deep-rooted perennial broadleaves such as Canada thistle, bindweed, leafy spurge and larkspur are readily controlled with 2 pounds per acre. White top (Lepedium sp.), peppercress and related species generally require 3 pounds per acre for good kills. Among woody plants, maple, cherry, aspen, cottonwood, birch, locust, rose, poison oak, and most conifers are quite susceptible to Tordon. Oak generally requires higher dosages for good kills and some species such as ash or toyon all show some resistance.

B. Phytotoxicity

Lee (129) reported that picloram adversely affected seed production in a number of grasses (e.g. Colonial bentgrass) in Oregon at rates of 1.0 to 1.5 pound per acre; however, none of treatments affected seedling development. Wheat (Triticum aestivum L.) was most susceptible to herbicide damage at the late tiller stage when 0.5 oz. of picloram per acre significantly reduced kernel yield (163). Both monocots in the seedling stage and dicots at all stages of development are adversely affected by low to moderate rates of picloram. However, at very low rates (e.g. 5 x 10-5 M) picloram promoted growth in soybeans. Baur et al. (18) reported that solutions of picloram at 0.25 to 0.50 ppb stimulated a significant increase in the fresh weight of corn, soybeans, cotton, cowpeas, and sorghum and wheat at 100 ppb. Picloram at 100 ppb caused a reduction in the fresh and dry weight of dicot species while a decrease was found in corn, wheat, and sorghum at 1,000 ppb. Rice was not affected at 1,000 ppb. Grover (92) reported that the effective dose (ED_{50}) which affected 50 percent of sunflower plants was not correlated to clay content when soil applications were used, but it was significantly related to soil organic matter. High ED50 values were required when pH was lowered or raised above 6.5. The lowest concentrations of picloram in ppm giving detectable symptoms in some of the more sensitive plants are as follows: pinto beans 0.02, pole beans, soybeans, safflower, and sunflower at 0.001 ppm (240).

Relatively low rates of Tordon may affect desirable plants, thus care in application should be exercised to avoid spray drift or contamination of irrigation water.

At equivalent rates of application, the phytotoxic effect of picloram may last longer than that of urea and triazine herbicides when crops sensitive to picloram are planted after its application. Included among the more sensitive crops are legumes, tomato, cucumber, potato, cotton, safflower, sunflower, lettuce, buckwheat, sugar beets, tobacco and soybeans. However, the phytotoxic action may not persist as long as for urea and triazine herbicides when lower rates of picloram are used, or when crops tolerant to picloram are planted after application of the herbicide.

C. Translocation

Evidence indicates that picloram is readily translocated throughout a plant and it is picked up both by the foliage and the roots. Sharma et al. (201) reported that canadian thistle (Cirsium arrense (L) Scop.) readily adsorbed picloram and translocated it in both the phloem and the xylem. It

tended to accumulate in young growing leaves where a substantial portion of it was retained. Small amounts of picloram were exuded by the roots into the soil. Isensee et al. (116) found that picloram was rapidly absorbed by oats and soybeans, with substantial redistribution in the plant and some exudation from the roots back into the culture medium. Picloram uptake decreased with an increase in pH from 3.5 to 4.5, but pH had little effect from 4.5 to 9.5. Low concentrations of metabolic inhibitors (e.g. 2,4-dinitrophenol, sodium arsenite) stimulated picloram translocation and high concentrations depressed it.

Tordon herbicide is absorbed and translocated readily by both roots and tops of most plants. It is moved to all parts of plants readily. In rapidly growing susceptible plants, symptoms of leaf and stem twisting may be visible in 1 to several hours after exposure. Later symptoms include leaf cupping, pointed leaves and fruit and epinasty similar to other growth regulator herbicides.

Tordon herbicide may be applied to plants as foliar sprays, soil treatments or trunk injections. Best results on deep rooted perennial broadleaf weeds are obtained when sprays are applied to the foliage before bloom and rain falls soon afterward. Applications can be made at any time of the year when action through soil is expected. Kills will not be effected, however, until the chemical is taken into the root zone. Spot treatments with Tordon in cropland are possible but food or feed crops should not be harvested from the treated area until residue tolerances have been established for this use.

D. Persistence in soil, water, or plants

In a review article on the movement and degradation of picloram, Goring and Hamaker (239) noted that it is broken down in plants, in the soil, and by pure cultures of microorganisms, and it can be degradated by sunlight. There is evidence the decomposition is most rapid in slightly acid soil. Leaching through the soil accounts for loss of a major amount of the picloram, especially in sandy soils in areas of high rainfall. However, it may not be readily leached out of the top 4 feet of heavy soils. Because of its low vapor pressure, loss by volatilization is negligible. There is evidence that only a small amount of picloram will be removed from an area in runoff water. Studies indicate that all of the picloram applied to soils cannot be accounted for, and further studies are needed to determine the fate of picloram in the environment (29).

1. Soils. Hamaker et al. (102) found that the percent of picloram decomposition was generally greater at lower initial concentrations. For practical purposes, half-order kinetics were more useful and almost as accurate as Michaelis-Menten kinetics for predicting the rate of picloram decomposition in soils. Half-order kinetics provide a useful relationship between the rate of decomposition and concentration. Results also suggest some correlation between soil organic matter content and the rate of herbicide decomposition. This is not surprising since other studies indicate that the maximum rate of decomposition is related to the activity of the microbial population which is in turn related to the amount of organic matter present (239). The proportion of ionized to non-ionized picloram decreases with decreasing soil pH. There is an increase in soil adsorption with decreasing pH and increasing organic matter content (92). Minimum adsorption occurs in neutral or alkaline sandy soils low in organic matter, and it increases with higher amounts of hydrated iron and aluminum oxides (100).

Hamaker et al. (241) estimated the rate of picloram breakdown in different climatic regions in the U.S.A. and determined that the half-order constants, $K_{1/2}$, vary from about 0.2 in colder, dryer areas to 1.0 in hotter, wetter areas. With initial rates of 1 oz. and 2 pounds a.e. per acre, the time for decomposition to concentration of 0.01 oz. per acre would take from 4.5 months to 4.6 years respectively, where the $K_{1/2}$ is 0.2 and from 0.9 to 11.0 months where the $K_{1/2}$ is 1.0. These predicted values were found to have good correlation with field data. Based on plots in California, South Dakota, Kansas, and Minnesota, the disappearance of picloram applied at rates of 1.4 to 4.2 pounds per acre ranged from 58 to 90 percent the first year and 78 to 100 percent the second year. The estimated half-life of picloram ranged from 1 to 13 months.

Bovey and Scifres (36) reviewed the literature concerning the residual characteristics of picloram in a grassland ecosystem and noted that most investigators agree that dissipation was accelerated at higher temperatures. Picloram was least persistent in sandy soils and loss is probably due to leaching. The soil pH and percent clay content did not affect the rate of decomposition while percent organic matter and moisture content and temperature were important.

Bovey and Scifres described the movement and loss of picloram through soil profiles in subhumid, tropical, and semiarid sites. In vegetated, subhumid areas, picloram at 2 pounds per acre disappeared from the top 2 feet of soil within 12 weeks and an 8-pound rate was not detectable in the top 2 feet a year after application. Only 10 to 25 percent of the applied picloram actually reached the soil surface. In another test, usually less than 2 ppb of picloram were found at all levels down to 8 feet one year after a 1-pound per acre application of the K-salt. On fallowed areas, soil texture, herbicide rate, and rainfall governed the degree and rate of vertical picloram movement. On vegetated tropical sites, only 5 ppb were detected one year after treatment with 9 pounds a.e. per acre. On all such locations there was rapid movement from the top 12 inches of soil and leaching was the most important means of picloram dissipation. The probability of sufficient rainfall for leaching in a semiarid site is obviously less than in a humid one; here photodecomposition is probably important. On slopes exceeding 3 to 4 percent, lateral leaching may be more important than available information indicates, especially following heavy rainfall.

The leaching pattern of picloram esters and salts are similar (36). Where esters were used, the unhydrolyzed esters were found in the top 5 cm of soil; only the acid form was found below 5 cm. It was not possible to distinguish between the acid and salt formulations. The salt form was not affected by temperature and it was less subject to photodecomposition than the ester form. Merkle et al. (146) found 15 to 25 percent of the picloram still present in soils even after applications as low as 0.5 pound per acre. The original soil moisture content did not affect the depth of leaching.

Bovey and Scifres (36) reported that little data is available to substantiate microbial breakdown of picloram in soils. They suggested that resistance to microbial degradation may account for its long persistence. Youngson et al. (239) studied the effect of 19 microorganisms on the decomposition of 1 ppm of picloram in nutrient cultures. Decomposition was small, ranging from 0.2 to 1.2 percent and picloram was not a preferred energy source by any of the test microorganisms. Approximately 10,000 to 100,000 pounds of organic matter would be broken down to each pound of picloram.

Merkle et al. (146) found that the effectiveness of soil applications of picloram was reduced if extended periods of hot, sunny weather followed which suggested that photodecomposition might result in a loss of picloram activity. Hall et al. (98) found that UV light caused a 20 percent degradation of picloram for each 48 hours of exposure. Decarboxylation did not appear to be a major pathway in photodecomposition. The possibility that degradation was by a free radical mechanism was considered plus the possibility of using inhibitors of free radical reactions to prevent photodecomposition. Merkle et al. (146) found that photodecomposition of picloram in petri dishes by UV light greatly exceeded that which occurred in the field, which were 90 and 15 percent respectively.

2. Water. Norris (165) reported on the presence of residues of summer-applied picloram in stream water in Oregon after the first fall storms. In an area where 67 percent of a watershed was sprayed in August, residues up to a maximum of 78 ppb were detected after the initial 1 inch storm and they decreased thereafter. No residues were found after late October or where only a small portion of a watershed was treated. In a chaparral area in southern California after an August application of 1, 2, and 4 pounds a.e. per acre of picloram, the first runoff water contained 0.1, 0.5, and more than 0.5 ppm of picloram respectively (90). After 15 inches of rain, residues had dropped to 0.01, 0.03, and 0.03 ppm of picloram.

   Haas et al. (97) reported that water that collected in ponds adjacent to treated areas contained picloram up to 184 ppb when runoff occurred within 2 weeks after application. Maximum picloram concentration was only 28 ppb if the first runoff did not occur until 6 weeks. Picloram concentration in pond water decreased rapidly the first 100 days down to a relatively stable concentration of ca. 5 ppb. It was not found in detectable concentration 0 or 0.5 miles downstream from an 80 acre area 5 months after an application of ca. 1 pound a.e. per acre, although the first runoff water contained up to 29 ppb. No picloram contamination was found in well water up to 2 years after adjacent areas were treated with ca. 1 pound a.e. per acre.

Various studies cited by Bovey and Scifres (36) indicated that water that runs off a treated area a few days after picloram treatment contained up to 184 ppb of picloram, but no residues were detected 6 months to 1 year later. However, the authors refer to a report (53) where a maximum of 370 ppb of picloram in water from an area treated 7 days before with 9 pounds per acre of picloram. Since there was a possible 22-fold dilution of picloram concentration in this test, the authors warned that crop damage could result from irrigation with water from treated watersheds. Picloram was present only in trace amounts in 3 months and it was undetectable within a year. Vegetative growth of sensitive crops would probably not be reduced by single irrigation with water containing 1 to 4 ppb of picloram, but a concentration of 10 ppb or more could severely affect some sensitive crop seedlings (36), Schneider et al. (192) reported that a sand aquifer accidentally contaminated by picloram would not be a hazard if the well was pumped soon after contamination, but if pumping was delayed several weeks, herbicide recovery would no longer be practical.

3. Plants. Interception of picloram sprays by vegetation would reduce the amount of picloram residue in the soil; a dense stand of oak might intercept up to 90 percent of the amount of picloram applied (147). The residual level of picloram in or on grass rapidly decreased after the initial deposit of liquid spray which amounted to a maximum of up to 200 ppm for each pound applied per acre (81). This decreased to less than 50 ppm in 2 weeks. An average of 91 percent of the picloram was gone by the next growing season and ranged from 60 percent in Montana to 100 percent in Georgia, Oklahoma, and Texas. Grass in the spring the following year after application showed no residue to a maximum of 12 ppm/pound/acre. No nonextractable residues (by normal extraction procedure) were found. Plant residues from granular formulations increased to a maximum at about 8 weeks after application, and they were generally lower than those found after foliar applications.

Baur and Bovey (15) found that grasses treated with picloram up to 2 pounds per acre contained an average of 2,650 ppb of fresh weight 1 month after treatment which dropped to 10 ppb in 6 months. Bovey and Scifres (36) referred to work in semiarid areas which indicated a 90 percent dissipation of picloram from grass 30 days after treatment. However, root uptake accounted for a delayed increase in picloram residue concentration. Accumulations like this were not found in the more humid areas where picloram was rapidly leached to the lower part of the soil profile.

Residues of picloram in woody plants in tropical areas ranged from 31 to 654 ppm immediately after spraying 2 pounds a.e. per acre of picloram to less than 1 ppm a month later (36). Live oak plants in subhumid areas dissipated 99 percent of the amount detected at 1 month, 6 months later. In semiarid areas (e.g. northwest Texas) picloram was reduced by 99 percent within 30 days after application to broadleaved species (199). Leaves from treated mesquite and chinnery oak increased the picloram content of the surface litter at 60 days compared to that 30 days after treatment.

E. Compatibility

Picloram has been formulated in various combinations with several of the phenoxy herbicides (see section I-E). Alley (5) reported that 2,4-D used in combination with low rates of picloram gave better control of deep-rooted perennial weeds with lower rates of picloram than when it was used alone. Interactions of picloram and phenoxy herbicides may be either additive or competitive based on plant response, but a certain amount of picloram was replaceable by phenoxies without reducing phytotoxicity (122). Meyer and Riley (150) found that mixing picloram with phenoxy herbicides, diesel oil, or ammonium thiocyannate did not increase whitebush control.

IV. Environmental impact

Some reference to the effect of picloram on plants and animals are made in previous sections.

A. Effects on nontarget organisms

In a review article, Bovey and Scifres (36) concluded that picloram residues do not appear harmful to mammals, fish, birds, or insects which inhabit the ecosystem. Picloram passes rapidly, intact through mammalian systems without apparent detrimental effects even at relatively high concentrations. Biological significance is related primarily to plant life.

Goring et al. (88) reported on the effect of picloram on microorganisms. Tests were run *in vitro* in both liquid and agar mediums which contained concentrations of picloram from 0 to 1,000 ppm. After 2 to 3 days growth and numbers were compared visually. Tests were also run *in vivo* on 50-gr. quantities of air dry soil treated with picloram at 0 to 1,000 ppm and incubated for 1 day to 1 month. Colonies were counted after 4 days and compared to those in soil to which only water was added. The results of studies with 46 different soil microorganisms indicated that concentrations up to 1,000 ppm did not retard the growth and development of any of the organisms except *Thiobacillus thiooxidans*. Nitrification of ammonium to nitrite while

partially inhibited at 1,000 ppm a.e. was not inhibited at 100 ppm; nitrification of nitrite to nitrate in soil was not inhibited at 1,000 ppm. Tu and Bollen (220) studied the effect of picloram on microorganisms in three Oregon soils and they also found that picloram had little obvious effect at concentrations up to 1,000 ppm on ammonification, nitrification, sulfur oxidation, and organic matter decomposition.

Reference to the effect of picloram on fish, birds, and other animals was made in section "III-A". McCollester and Leng (143) estimated the acceptable daily intake of picloram for man, based on extrapolation of long and short-term toxicity studies in laboratory animals. Based on the procedures established by the Joint FAO/WHO Expert Committee on Food Additives and employing a 100-fold safety margin, the acceptable daily intake of picloram for man is calculated to be 1.5 mg/kg of body weight per day. Assuming that a person is in the top 10 percent of consumers whose food consumption is 1.5 to 3.5 times the mean for broad groups in the United States, a person would consume only 0.1 mg per day from meat of animals grazed continuously on grasses containing 200 to 400 ppm of picloram. This is only a fraction of the 90 mg/day that a 130 pound man could safely consume, and it represents a safety margin of 90,000 to 1 compared to the no ill effect level demonstrated in laboratory animals.

B. Residues in or on food or feed or entering the food chain

Reference to residues and persistence of picloram in soil, water, and plants was made in section "III-D". MacLean and Davidson ( ) who referred to the toxilogical work by Palmer and Radeleff (167) noted that assuming a given amount of forage yield, forage consumption, and that all chemical applied sticks to the vegetation, a maximum dosage possible would be 7 mg/kg for each pound per acre of herbicide applied. Therefore, the maximum dosage that cattle might ingest if fed on vegetation immediately after spraying 4 pounds per acre of picloram would be 28 mg/kg.

Work by Kutschinski (124) on residues in milk from cows fed daily rations containing picloram at rates up to 1,000 ppm, equivalent to 18 mg/kg/day for 2 weeks, showed residues from 0.05 to 2.0 ppm. The residue levels dropped to less than 0.02 ppm within 2 to 3 days after withdrawal. In a similar test, tissue of steers fed up to 1,600 ppm in their total diet (equivalent to 23 mg/kg/day) reached a maximum in the blood after 3 days of feeding and rapidly declined after withdrawal (125). During this time, the residues were less than 0.05 to 5.0 ppm in muscle and fat, 0.1 to 2.0 ppm in blood and liver, and 2 to 18 ppm in kidney. They decreased to less than 0.1 in kidney and less than 0.05 ppm in other tissue 3 days after withdrawal. Concentrations of 200 to 400 ppm of picloram were required in the diet of cattle to produce residues of 0.05 to 1.0 ppm in edible tissue such as fat and muscle.

BIBLIOGRAPHY OF PICLORAM REFERENCES

(Most Weed Society Proceedings and governmental publications have not been included)

1. Agbakoba, Chuma S.O. and J.R. Goodin. 1969. Effect of stage of growth of field bindweed on absorption and translocation of ^{14}C-labeled 2,4-D and picloram. Weed Sci. 17(4): 436-438.

2. Agbakoba, Chuma S.O. and J.R. Goodin. 1970. Absorption and translocation of ^{24}C-labeled 2,4-D and picloram in field bindweed. Weed Science 18(1): 168-170.

3. Agbakoba, Chuma S.O. and J.R. Goodin. 1970. Picloram enhances 2,4-D movement in field bindweed. Weed Science 18(1): 19-21.

4. Alley, H.P. 1965. A promising future for the control of perennial weeds. Down to Earth 21 (1-2): 8-10.

5. Alley, H.P. 1967. Some observations on tordon-2,4-D herbicide combinations. Down to Earth 23(1): 2, 35-36.

6. Alley, H.P. and G.A. Lee. 1966. Crop tolerance to picloram residuel. West. Weed Contr. Conf. Res. Rep. p. 102

7. Aly, O.M. and S.D. Faust. 1964. Studies on the fate of 2,4-D and ester derivatives in natural surface water. J. Agr. Food Chem. 12: 541-546.

8. Arnold, W.R., P. W. Santelmann, and J.Q. Lynd. 1966. Picloram and 2,4-D effects with Aspergillus niger proliferation. Weeds 14: 89-90.

9. Arvik, J.H., D.L. Willson, and L.C. Darlington. 1971. Response of soil algae to picloram - 2,4-D mixtures. Weed Science 19(3): 276-278.

10. Ashton, F.M., D. Penner, and S. Hoffman. 1968. Effect of several herbicides on proteolytic activity of squash seedlings. Weed Sci. 16(2): 169-171

11. Bachelard, E.P. and R.D. Ayling. 1971. The effect of picloram and 2,4-D on plant cell membranes. Weed Res. 11(1): 31-36.

12. Bachelard, E.P. and V.H. Boughton. 1967. The effect of weedicides on growth of radiata pine seedlings. Aust. Forest 31(3): 211-220.

13. Bachelard, E.P. and R. Sands. 1966. Effect of weedicides on starch content and coppicing of cut stumps of manna gum. Aust. Forest 32(1): 49-54.

14. Balayannis, P.G., M.S.Smith, and R.L. Wain. 1965. Studies on plant growth regulating substances. XX. The metabolism of V-(2,4,5-trichlorophenoxy) butyric acid in what and pea stem tissues. Annals of Applied Biology 55: 261-265.

15. Baur, J.R. and R.W. Bovey. 1969. Distribution of root-absorbed picloram. Wed Sci. 17(4): 524-528.

16. Baur, J.R. and R.W. Bovey. 1970. The uptake of picloram by potato tuber tissue. Weed Science 18(1): 22-24.

17. Baur, J.R., R.W. Bovey, R.D. Baker, and I. Riley. 1971. Absorption and penetration of picloram and 2,4,5-T into detached live oak leaves. Weed Science 19(2): 138-141.

18. Baur, J.R., R.W. Rovey, and C.R. Benedict. 1970. Effect of picloram on growth and protein levels in herbaceous plants. Agron. J. 62:627-630.

19. Baur, J.R., R.W. Bovey, and J.D. Smith. 1969. Herbicide concentrations in live oak treated with mixtures of picloram and 2,4,5-T. Weed Sci. 17(4): 567-570.

20. Baur, J.R. and P.W. Morgan. 1969 Effects of picloram and ethylene on leaf movement in Huisache and Mesquite seedlings. Plant Physiol. 44(6): 831-838.

21. Beger, H.W. 1970. Treatment of bloodwood (Fucalypus dichromophloia) with picloram/2,4-D and the pentrant dimethyl sulphoxide. Queens J. Agar. Anim. Sci. 27(1): 17-20.

22. Biswas, P.K. and R.L. Haynes. 1970. Herbicidal effects on water uptake of seeds of selected weed species. Physiol. Plant 23(3); 583-590.

23. Bjerke, E.L., A.H. Kutschinski, and J. C. Ramsey. 1967. Determination of residues of 4-amino-3,5,6-trichloropicolinic acid in cereal grains by gas chromatography. J. Agr. Food Chem. 15: 469-473.

24. Bovey, R.W. 1969. Effects of foliarly applied desiccants on selected species under tropical environment. Weed Sci. 17(1): 79-83.,

25. Bovey, R.W., J. R. Baur, and H.L. Morton. 1970. Control of huisache and associated woody species in South Texas. J. Range Manage. 23:47-50.

26. Bovey, R.W., F.S. Davis, and M.G. Merkle. 1967. Distribution of picloram in Huisache after foliar and soil applications. Weeds 15(3): 245-249.

27. Bovey, R.W. and J. D. Diaz-Colon. 1969. Effect of simulated rainfall on herbicide performance. Weed Sci. 17(2): 154-157.

28. Bovey, R.W., C.C. Dowler, and J. C. Diaz-Colon. 1969. Response of tropical vegetation to herbicides. Weed Sci. 17(3): 285-290.

29. Bovey, R.W., C.C. Dowler, and M.G. Merkle. 1969. The persistence and movement of picloram in Texas and Puerto Rican soils. Pest. Monit. J. 3:177-181.

30. Bovey, R.W., M.L. Ketchersid, and M.G. Merkle. 1970. Comparison of salt and ester formulations of picloram. Weed Science 18(4): 447-451.

31. Bovey, R.W., R.E. Meyer, F.S. Davis, M.G. Merkle, and H.L. Morton. 1967. Control of woody and herbaceous vegetation with soil sterilants. Weeds 15(4): 327-330.

32. Bovey, R.W. and F.R. Miller. 1969. Effect of activated carbon on the phytotoxicity of herbicides in a tropical soil. Weed Sci. 17(2): 189-192.

33. Bovey, R.W., F.R. Miller, and J. Diaz-Colon. 1968. Growth of crops in soils after herbicidal treatments for brush control in the tropics. Agron. J. 60:678-679.

34. Bovey, R.W., H.L. Morton, and J.R. Baur. 1969. Control of live oak by herbicides applied at various rates and dates. Weed Sci. 17(3): 373-376.

35. Bovey, R.W. H.L. Morton, J.R. Baur, J.D. Diaz-Colon, C.C. Dowler, and S.K. Lehman. 1969. Granular herbicides for woody plant control. Weed Sci. 17(4): 538-541.

36. Bovey, R.W. and C.J. Scifres. 1971. Residual characteristics of picloram in grassland ecosystems. Tex. Agr. Exp. Sta. B-1111. 24 p.

37. Brender, E.V. and E.L. Moyer. 1965. Further progress in the control of Kudzu. Down to Earch 20(4): 16-17.

38. Burnside, O.C., G.A. Wicks, and C.R. Fenster. 1971. Dissipation of dicamba, picloram, and 2,3,6-TBA across Nebraska. Weed Science 19(4): 323-325.

39. Butts, J.S. and S.C. Fang. 1956. Tracer studies on the mechanism of action of hormonal herbicides. U.S. Atomic Energy Rpt. TID 7512, p. 209-214.

40. Byrd, B.C. and F.A. Nyman, Jr. 1966. Progress report on highway vegetation control experiments using tordon 101 mixture and norbak particulating agent. Down to Earth 22(1): 28-31.

41. Carvell, K.L. 1968. Tordon effective in red maple tree injection studies. Down to Earth 24(3): 17-18.

42. Chang, In-Kook and C.L. Foy. 1971. Effects of picloram on germination and seedling development of four species. Weed Science 19(1): 58-64.

43. Chang, In-Kook and C.L. Foy. 1971. Effects of picloram on mitochondrial swelling and ATPase. Weed Science 19(1): 54--57.

44. Cheng, H.H. 1969. Extraction and colorimetric determination of picloram in soil. J. Agr. Food Chem. 17(6): 1174-1177.

45. Coble, H.D., R.P. Upchurch, and J.A. Keaton. 1969. Influence of time and method of application on turkey oak response to picloram plus 2,4-D. Weed Sci. 17(1): 87-91.

46. Coble, H.D., R.P. Upchurch, and J.A. Keaton. 1969. Response of woody species to 2,4-D, 2,4,5-T, and picloram as a function of treatment method. Weed Sci. 17(1): 40-46.

47. Corbin, F.T. and R.P. Upchurch. 1967. Influence of pH on detoxication of herbicides in soil. Weeds 15(4): 370-377.

48. Corbin, F.T., R.P. Upchurch, and F.L. Selman. 1971. Influence of pH on the phytotoxicity of herbicides in soil. Weed Science 19(3): 233-239.

49. Corns, W.G. 1967. Effects of added surfactant on toxicity of picloram, 2,4-D and 2,4,5-T to Populus tremuloides Michx, and P. balsamifera L. saplings. Can. J. Plant Sci. 47(6): 711-712.

50. Couch, R.W. and D.E. Davis. 1966. Kudzu control with tordon. Down to Earth 22(3): 2.

51. Cozart, E.R. 1965. Reclamation of Macartney rose infested fence rows with tordon herbicide. Down to Earch 21 (1-2): 15-18.

52. Dana, M.N. 1967. Brush control in sphagnum moss bogs. Weeds 15(4): 380-381.

53. Davis, E.A., P.A. Ingebo and C.P. Pase. 1968. Effect of a watershed treatment with picloram on water quality. USDA, Forest Service Research Note RM-100. p.4.

54. Davis, F.S., R.W. Bovey, and M.G. Merkle. 1968. The role of light, concentration, and species in foliar uptake of herbicides in woody plants. Forest Sci. 14: 164-169.

55. Dexter, A.G., F.W. Slife, and H.S. Butler. 1971. Detoxification of 2,4-D by several plant species. Weed Science 19(6): 721-726.

56. Dickens, R. and G.A. Buchanan. 1971. Influence of time of herbicide application on control of kudzu. Weed Science 19(6): 669-671.

57. Dos Santos, C.A. Lobato, and N. Grassl. 1970. Extermination of "lieteiro" (Tabernaemontana fuchsiaefolia D.C.) with herbicides. Biologico (Sao Paulo) 36(10): 280-283.

58. Dowler, C.C. 1969. A cucumber bioassay test for the soil residues of certain herbicides. Weed Sci. 17(3): 309-310.

59. Dowler, C.C., W. Forestier, and F.H. Tschirley. 1968. Effect and persistence of herbicides applied to soil in Puerto Rican forests. Weed Sci. 16(1): 45-50.

60. Dowler, C.C. and F.H. Tschirley. 1970. Evaluation of herbicides applied to foliage of four tropical woody species. J. Agr. Univ. P.R. 54(4): 676-682.

61. Dowler, C.C., F.H. Tschirley, R.W. Bovey, and H.L. Morton. 1970. Effect of aerially-applied herbicides on Texas and Puerto Rico forests. Weed Science 18(1): 164-168.

62. Dubey, H.D. 1969. Effect of picloram, diuron, ametryne, and prometryne on nitrification in some tropical soils. Soil Sci. Soc. American Proc. 33(6): 893-896.

63. Edmond, D.B. and E.M. Wright. 1964. The effect of 4-amino-3,5,6-trichloropicolinic acid on ryegrass-white clover pastures. New Zeal. J. Agr. Res. 7(4): 770-773.

64. Egley, G.H. and J.E. Dale. 1970. Ethylene, 2-chloroethylphosphonic acid and witchweed germination. Weed Science 18(5): 586-589.

65. Elsinger, W. and D.J. Moore. 1965. Tordon a new synthetic growth regulator. Proc. Indiana Acad. Sci. 75: 63 (Abstract only).

66. Elsinger, W.R. and D.J. Morre. 1971. Growth regulating properties of picloram 4-amino-3,5,6-trichloropicolinic acid. Can. J. Bot. 49(6): 889-897.

67. Elsinger, W., D.J. Morre, and C.E. Hess. 1966. Promotion of plant growth by tordon herbicide. Down to Earth 21(4): 8-10.

68. Elwell, H.M. 1968. Winger elm control with picloram and 2,4,5-T with and without additives. Weed Sci. 16(2): 131-133.

69. Fenster, C.R., O.C. Burnside, and G.A. Wicks. 1966. Comparison of the residual effects of dicamba, picloram, and 2,3,6-TBA with field beans (Phaseolus vulgaris L.). Proc. North Cent. Weed Cont. Conf. 20:20

70. Ferguson, R.H. 1965. Tordon controls range brush in New Zealand and Australia. Down to Earth 21(3): 18-21.

71. Finnis, J.M. 1967. The effect of tordon on vine maple. Down to Earth 22(4): 22-23.

72. Finnis, J.M. and J.D. Sund. 1970. Planting of Douglas-fir seedlings following aerial application of tordon 101 mixture herbicide. Down to Earth 26(1): 10-11.

73. Fisher, D.A., D.E. Bayer, and T.E. Weier. 1968. Morphological and anatomical effects of picloram on Phaseolus vulgaris. Bot. Gaz. 129(1): 67-70.

74. Fisher, C.E. E.D. Robison, G.O. Hoffman, C.H. Meadors, and B.T. Cross. 1970. Aerial application of chemicals for control of brush on rangeland. Brush Research in Texas/1970, Texas A&M Univ., Texas Agr. Exp. Sta., College Station. p. 5-11. PR-2801.

75. Fisher, D.E., L.E. St. John, Jr., W.H. Gutermann, D.G. Wagner, and D.J. Lisk. (Pesticide Residue Lab., Cornell Univ., Ithaca, N.Y., USA) Fate of Banvel T, loxynii, tordon, and trifluorilin in the dairy cow. J. Dairy Sci. 48(12): 1711-1715.

76. Fletcher, J.T. 1968. The (phytotoxic) effect of picloram on tomatoes and cucumbers. Weed Res. 8(2): 153-155.

77. Foote, L.E., D.L. Kill, and C. S. Williams. 1970. Canada thistle control on roadsides. Down to Earch 26(2): 22-26.

78. Friesen, G. 1965. Wild buckwheat control with tordon. Down to Earch 20(4): 9-10.

79. Friesen, H.A., and D.A. Dew. 1966. (Exp. Farm, Can. Dep. Agr., Lacombe, Alberta Can.) The influence of temperature and soil moisture on the phytotoxicity of dicamba, picloram, bromoxynil, and 2,4-D ester. Can. J. Plant Sci. 46(6): 653-660.

80. Gantz, R.L. and L.E. Warren. 1966. Wild buckwheat control in small grain crops with tordon herbicide. Down to Earth 22(1): 13-15.

81. Getzenduner, M.E., J.L. Herman, and Bart VanGlessen. 1969. Residues of 4-amino-3,5,6-trichloropicolinic acid in grass from application of Tordon herbicides. Agr. and Food Chem. 17(6): 1251-1256.

82. Gibson, J.W. 1969. Weed control with repeated applications of tordon herbicides. Down to Earch 25(3): 12-14.

83. Gibson, J. W. and J.B. Grumbles. 1970. Aerial application of herbicides for control of whitebrush and associated species. Down to Earth 26(2): 1-4.

84. Goldschmidt, E.E. and B. Leshem. 1971. Style abscission in the citron (Citrus medica L.) and other citrus species: Morphology, physiology and chemical control with picloram. Amer. J. Bot. 58(1): 14-23.

85. Goodin, J.R. and F.L.A. Becher. 1967. Picloram as an auxin substitute in tissue culture. Plant Physiol. 42:523.

86. Goodin, J.R. and Wei-Chin Chang. 1969. A new selective bioassay for tordon in water. Down to Earth 24(4): 4-5.

87. Goodin, J.R. L.S. Jordan, and W.H. Isom. 1967. Low rates of tordon for field bindweed control. Down to Earth 22(4): 6-7.

88. Goring, C.A.I., J.D. Griffith, F.C. O'Melia, H.H. Scott, and C.R. Youngson. 1967. The effect of tordon on microorganisms and soil biological processes. Down to Earth 22(4): 14-17.

89. Goring, C.A.I., C.R. Youngson, and J.W. Hamaker. 1965. Tordon herbicide...disappearance from soils. Down to Earth 20(4): 3-5.

90. Green, Lisle R. 1970. Effect of picloram and phenoxy herbicides in small chaparral watersheds. Res. Progress Rpt., Western Soc, of Weed Science, Sacramento, p. 24-25.

91. Grover, R. 1967. Studies on the degradation of 4-amino-3,5,6-trichloropicolinic acid in soil. Weed Res. 7:61-67.

92. Grover, R. 1968. Influence of soil properties on phytotoxicity of 4-amino-3,5,6-trichloropicolinic acid (picloram). Weed Res. 8:226-232.

93. Grover, R.1970. Influence of soil-moisture content on the bioactivity of picloram. Weed Science 18(1): 110-111

94. Grover, R. 1971. Adsorption of picloram by soil colloids and various other adsorbents. Weed Science 19(4): 417-418.

95. Guazzelli, R.J. and G.P. Rios. 1966. Herbicide test on Mata-Barata (Andira sp.). Pesquis Agropecuar, Brasil 1: 329-332.

96. Haagsma, T. and E.E. Wiffen. 1966. Farm evaluation of tordon plus phenoxy herbicide combination for weed control in spring wheat in western Canada--1965. Down to Earth 21(4): 22-23.

97. Haas, R.H., C.J. Scifres, M.G. Merkle, R.R. Hahn, and G.O. Hoffman. 1971. Occurrence and persistence of picloram in natural water resources. Weed Res. 11:54-62.

98. Hall, R.C., C.S. Giam, and M.G. Merkle. 1968. The photolytic degradation of picloram. Weed Res. 8:292-297.

99. Hall, R.C., C.S. Giam, and M.G. Merkle. 1970. A new technique for the determination of picloram and other herbicides containing carboxylic acid and ester groups. Analytical Chem. 42: 423-425.

100. Hamaker, J.W., C.A.I. Goring, and C.R. Youngson. 1966. Sorption and leaching of 4-amino-3,5,6-trichlorcpicolinic acid in soils. Adv. Chem. Ser., 60:23-27.

101. Hamaker, J.W., H.Johnston, R.T. Martin, and C.T. Redemann. 1963. A picolinic acid derivative: A plant growth regulator. Science, N.Y., 141-363.

102. Hamaker, J.W., C.R. Youngson, and C.A.I. Goring. 1968. Rate of detoxification of 4-amino-3,5,6-trichloropicolinic acid in soil. Weed Res. 8:46-57.

103. Hance, R.J. 1969. Further observations of the decomposition of herbicides in soil. J. Sci. Food Agr. 20(3): 144-145.

104. Hardy, J.L. 1966. Effect of tordon herbicides on aquatic chain organisms. Down to Earth 22(2): 11-13.

105. Harrigan, G. 1970. Chemicals answer northern Australian challenge. Down to Earth 26(3): 16-18.

106. Hart, G.L. 1966. Control of poison oak on military reservations with tordon. Down to Earth 22(3): 6-7.

107. Heikes, E.E. 1964. Tordon and other herbicides...field testing for the control of deep-rooted perennial weeds in Colorado. Down to Earth 20(3): 9-12.

108. Helling, C.S., D.D. Kaufman, and C.T. Dieter. 1971. Algae bioassay detection of pesticide mobility in soils. Weed Science 19(6): 685-690.

109. Hemphill, D.D. 1968. Performance of vegetable crops on an area treated with tordon herbicide. Down to Earth 24(1): 2, 24.

110. Herr, D.E., E.W. Stroube, and D.A. Ray. 1966. Effect of tordon residues on agronomic crops. Down to Earth 21(4): 17-18.

111. Herr, D.E., E.W. Stroube, and D.A. Ray. 1966. The movement and persistence of picloram in soil. Weeds 14(3): 248-250.

112. Hoffman, G.O. 1967. Controlling pricklypear in Texas. Down to Earth 23(1): 9-12.

113. Hoffman, G.O. 1971. Practical use of tordon 225 mixture herbicide on Texas rangelands. Down to Earth 27(2): 17-21.

114. Horton, R.F. and R.A. Fletcher. 1968. Transport of auxin, picloram, through petioles of bean and coleus and stem sections of pea. Plant Physiol. 43(12): 2045-2048.

115. Hull, H.M. and H.L. Morton. 1971. Morphological response of two mesquite varieties to 2,4,5-T and picloram. Weed Science 19(6): 712-716.

116. Isensee, A.R., G.E. Jones, and B.C. Turner. 1971. Root adsorption and translocation of picloram by oats and soybeans. Weed Science 19(6): 727-731

117. Kefford, N.P. and O. H. Caso. 1966. A potent auxin with unique chemical structure--4-amino-3,5,6-trichloropicolinic acid. Bot. Gaz. 127:159-163.

118. Kenaga, E.E. 1969. Tordon herbicides--evaluation of safety to fish and birds. Down to Earth 25(1): 5-9.

119. Klingman, G.C. and H. Guedez. 1967. Picloram and its effect on field-grown tobacco. Weeds 15(2): 142-146.

120. Kozlowski, T.T. and Sasaki. 1968. Germination and morphology of red pine seeds and seedlings in contact with EPTC, EDEC, CDAA, 2,4-D and picloram. Proc. Amer. Soc. Hort. Sci. 93:655-662.

121. Kozlowski, T.T., S. Sasaki, and J.H. Torrie. 1967. Effects of temperature on phytotoxicity of monuron, picloram, CDEC, EPTC, CDAA, and sesone to young pine seedlings. Silva Fenn. 1(3): 13-28.

122. Krawiec, S. and D.J. Morre. 1968. Interactions of tordon herbicide applied in combinations. Down to Earth 24(3): 7-10.

123. Kreps, L.B. and H.P. Alley. 1967. Histological abnormalities induced by picloram on Canada thistle roots. Weeds 15(1): 56-59.

124. Kutschinski, A.H. 1969. Residues in milk from cows fed 4-amino-3,5,6-trichloropicolinic acid. J. Agr. Food Chem. 17:288-290.

125. Kutschinski, A.H. and Van Riley. 1969. Residues in various tissues of steers fed 4-amino-3,5,6-trichloropicolinic acid. J. Agr. Food Chem. 17:283-287.

126. Laning, E.R., Jr. 1963. Tordon...for the control of deep-rooted perennial herbaceious weeds in the Western States. Down to Earth 19(1): 3-5.

127. Lawson, H.M. 1965. Chemical control of bracken fern in the British Isles with tordon. Down to Earth 20(4): 13-15.

128. Lee, G.A., A.K. Dobrenz, and H.P. Alley. 1967. Preliminary investigations of the effect of tordon and 2,4-D on leaf and root tissue of Canada thistle. Down to Earth 23(2): 21-23.

129. Lee, W.O. 1970. Effect of picloram on production and quality of seed in several grasses. Weed Science 18(1): 171-173.

130. Leiderman, L. and N. Grassl. 1970. Chemical control of the equatic plant aquape (water hyacinth) in the Rio Preto, municipality of Peruibe, Sao Paulo. Biologico (Sao Paulo) 36(6): 157-159.

131. Leonard, O.A., C.E. Carlson, and D.E. Bayer. 1965. Studies on the cut surface method. 11. Control of blue oak and madrone. Weeds 13(4): 352-356.

132. Leonard, O.A., R.K. Glenn, and D.E. Bayer. 1965. Studies on the cut-surface method. I. Translocation in blue oak and madrone. Weeds 13(4): 346-351.

133. Leonard, O.A., R.J. Weaver, and R.H. Glen. 1967. Effect of 2,4-D and picloram on translocation of ^{14}C-assimilates in Vitis vinifera L. Weed Res. 7(3): 208-219.

134. Lynn, G.E. 1965. A review of toxicological information on tordon herbicides. Down to Earth 20(4): 6-8.

135. MacRae, I.C. and M. Alexander. 1965. Microbial degradation of selected herbicides in soil. J. Agr. Food Chem. 13:72-76.

136. Malhotra, S.S. 1966. Aberrations of the nucleic acid metabolism of plants induced by 4-amino-3,5,6-trichloropicolinic acid. Ph.D. Diss., University of Illinois, Urbana, Illinois.

137. Malhotra, S.S. and J. B. Hanson. 1970. Picloram sensitivity and nucleic acids in plants. Weed Science 18(1): 1-4.

138. Mann, J.D. and M. Pu. 1968. Inhibition of lipid synthesis by certain herbicides. Weed Sci. 16(2): 197-198.

139. Marquiss, R.W. 1971. Gambel oak control studies. Down to Earth 27(2): 22-24.

140. Martin, S.C., S.J. Shellhorn, and H.M. Hull. 1970. Emergence of fourwing saltbush after spraying shrubs with picloram. Weed Science 18(3); 389-392.

141. McCarty, M.K. and C.J. Scifres. 1968. Smooth bromegrass response to herbicides as affected by time of application in relation to nitrogen fertilization. Weed Sci. 16(4): 443-446.

142. McCarty, M.K. and C.J. Scifres. 1969. Herbicidal control of western ironweed. Weed Sci. 17(1): 77-79.

143. McCollister, D.D. and M.L. Leng. 1969. Toxicology of picloram and safety evaluation of tordon herbicides. Down to Earth 25(2): 5-10.

144. Meikle, R.W., E.A. Williams, and C.T. Redemann. 1965. The synthesis of 4-amino-3,5,6-trichloropicolonic-carboxy C^{-14} acid and its use in a study of the metabolism of tordon herbicide in carbon dioxide evolution from treated soil. 149th Amer. Chemical Soc. Mtg., Abstracts, Detroit, Mich. April 4-9, p. 19A.

145. Menzie, C.M. 1966. Metabolism of pesticides. U.S. Fish and Wildlife Service. Special Scientific Rpt.--Wildlife No. 96. Patuxent Wildlife Res. Center, Laurel, Maryland. 274 p.

146. Merkle, M.G., R.W. Bovey, and F.S. Davis. 1967. Factors affecting the persistence of picloram in soil. Agron. J. 59:413-414.

147. Merkle, M.G., R.W. Bovey, and R. Hall. 1966. The determination of picloram residues in soil using gas chromatography. Weeds 14(2): 161-164.

148. Merkle, M. G. and F. S. Davis. 1967. Effect of moisture stress on absorption and movement of picloram and 2,4,5-T in beans. Weeds 15(1): 10-12.

149. Meyer, R.E. 1970. Picloram and 2,4,5-T influence on honey mesquite morphology. Weed Science 18(4): 525-531.

150. Meyer, R.E. and T.E. Riley. 1969. Influence of picloram granules and sprays on whitebrush. Weed Sci. 17(3): 293-295.

151. Mitich, L. W. 1966. Leafy spurge--a problem weed controlled by tordon. Down to Earth 21(4): 11-13.

152. Mitich. L.W. 1967. Control of leafy spurge, field bindweed and western snowberry with tordon herbicide. Down to Earth 23(3): 8-11.

153. Moffat, R.W. 1968. Some factors affecting the disappearance of tordon in soil. Down to Earth 23(4): 6-10.

154. Molberg, E.S. 1965. Experiments with tordon for weed control in flax. Down to Earth 20(4): 11-12.

155. Montaldi, E.R. 1970. Cynodon dactylon: A possible cause of its diageotropism. Rev invest agropecuar. Scr 2 Biol. Prod. Veg. 7(2): 67-87.

156. Moreland, D.E., S.S. Malhotra, R.D. Gruenhagen, and E.H. Shokraii. 1969. Effects of herbicides on RNA and protein. Weed Sci. 17(4): 556-563.

157. Morgan, P.W. and J.R. Baur. 1970. Involvement of ethylene in picloram-induced leaf movement responses. Plant Physiol. 46(5): 655-659

158. Morgan, P.W., R.E. Meyer, and M.G. Merkle. 1969. Chemical stimulation of ethylene evolution and bud growth. Weed Sci. 17(3): 353-355.

159. Moseman, R.F. and W.A. Aue. 1970. Novel determination of picloram by gas-liquid chromatography. Chromatogr. 49(3): 432-441.

160. Motooka, P.S., D.F. Saiki, D.L. Plucknett, O.R. Younge, and R.E. Daehler. 1967. Control of Hawaiian jungle with aerially applied herbicide. Down to Earth 23(1): 18-22.

161. Mounat, A., P. Schiltz, and F. Casamajour. 1968. Effects of picloram on tobacco. Seita Serv. Exploin Ind. Tabaca Allumettes Ann. Dir. Etud. Equip. Sect. 2 5: 67-74.

162. Nalewaja, J.D. 1970. Reaction of wheat to picloram. Weed Science 18(2): 276-278.

163. Nalewaja, J.D. and R. E. Bothun. 1969. Response of flax to postemergence herbicides. Crop Sci. 9(2): 160-162.

164. Nation, H.A. 1967. Report on tree control via injection with tordon 101 mixture. Down to Earth 23(2): 24-27.

165. Norris, L.A. 1969. Herbicide runoff from forest lands sprayed in summer. Res. Progress Rpt., Western Soc. of Weed Science, Las Vegas p 24-26.

166. Norris, L.A. 1969. Degradation of several herbicides in red alder forest floor material. Res. Progress Rpt., Western Soc. of Weed Science, Las Vegas, p. 21-22

167. Palmer, J.S. and R.D. Radeleff. 1969. The toxicity of some organic herbicides to cattle, sheep, and chickens. USDA Prod. Res. Rpt. No. 106.

168. Perala, D.A. and C.S. Williams. 1970. Site preparation for conifers. using herbicides and subsequent burning in a northern Minnesota hardwood stand. Down to Earth 26(3): 5-8.

169. Perry, P. W. and R. P. Upchurch. 1968. Growth analysis of red maple and white ash seedlings treated with eight herbicides. Weed Sci. 16(1): 32-37.

170. Peters, E.J. and S.A. Lowance. 199. Gains in Timothy forage from goldenrod control with 2,4-D, 2,4-DB, and picloram. Weed Sci. 17(4): 476-474

171. Peters, E.J. and J.F. Stritzke. 1970. Control of persimmon with various herbicides and methods of application. Weed Science 18(5): 572-575.

172. Plumb, T.R. 1968. Control of brush regrowth in southern California with tordon and phenoxy herbicides. Down to Earth 24(3): 19-22.

173. Plumb, T.R. 1971. Broadcast applications of herbicides to control scrub oak regrowth. USDA, Forest Serv. Pacific Southwest Forest & Range Experiment Sta., Berkeley, Calif. Res. Note PSW-261, 4 p., illus.

174. Pridham, A.M.S. and R.A. Schwartzbeck. 1965. Tordon herbicide for control of Japanese bamboo in the northeastern United States. Down to Earth 21(1-2): 21-22.

175. Rahman, A. and W.G. Corns. 1970. Response of winter rye to applications of 2,4-D and picloram at various growth stages. Can. J. Plant Sci. 50(5): 600-602.

176. Reber, L.J., R.K. Miller, J.A. Tweedy, and J.D. Butler. 1971. Herbicidal effects of picloram on bermudagrass. Weed Science 19(5): 521-524.

177. Redemann, C.T. 1965. Fate of 4-amino-3,5,6-trichloropicolinic acid in the rat. 150th Meeting, ACS, Atlantic City, Sept.

178. Redemann, C.T. 1965. Fate of 4-amino-3,5,6-trichloropicolinic acid in the dog. 150th. Meeting, ACS, Atlantic City, Sept.

179. Redemann, C.T., P.M. Hamilton, and C.R. Younger. 1965. The fate of 4-amino-3,5,6-trichloropyridine-2,3,4,5,6-C^{14}_5-2-carboxy-C^{14} acid in spring wheat. Abstracts of the 149th Amer. Chemical Soc. Mtg., Detroit, Mich., April 4-9: 20A-21A.

180. Redemann, C.T., R.W. Meikle, P. Hamilton, V.S. Banks, and C.R. Youngson. 1968. The fate of 4-amino-3,5,6-trichloropicolinic acid in spring wheat and soil. Bull. Env. Contamination and Toxicology 3(2): 80-82.

181. Reid, C.P.P. and W. Hurtt. 1969. A rapid bioassay for stimultaneous identification and quantitation of picloram in aqueous solution. Weed Res. 9(2): 136-141.

182. Reid, C.P.P. and W. Hurtt. 1969. Translocation and distribution of picloram in bean plants associated with nastic movements. Plant Physiol. 44(10): 1393-1396.

183. Reid, C.P.P. and W. Hurtt. 1970. Root permeability as affected by picloram and other chemicals. Physiol. Plant 23(1): 124-130.

184. Reimer, C.A., B.C. Byrd, and J.H. Davidson. 1966. An improved helicopter system for the aerial application of sprays containing tordon 101 mixture particulated with Norbak. Down to Earth 22(1): 3-6.

185. Renney, A.J. and E.C. Hughes. 1969. Control of knapweed, Centaurea species, in British Columbia with tordon herbicide. Down to Earth 24(4): 6-8.

186. Robison, E.D. 1967. Response of mesquite to 2,4,5-T, picloram, and 2,4,5-T picloram conbinations. Proc. So. Weed Sci. Soc. 20:199.

187. Saha, J.G. and L.A. Gadallah, 1967. Determination of the herbicide tordon (4-amino-3,5,6-trichloropicolinic acid) in soil by electron capture gas chromatography. Ass. Office Anal. Chem. J. 50(3): 637-641.

188. Sargent, J.A. and G.E. Blackman. 1970. Studies on foliar penetration. VI. Factors controlling the penetration of 4-amino-3,5,6-trichloropicolinic acid (picloram) into the leaves of Phaseolus vulgaris. J. Exp. Bot. 21(66): 219-227.

189. Sawamura, S. and W.T. Jackson. 1968. Cytological studies in vivo of picloram, pyriclor, trifluralin, 2,3,6-TBA, 2,3,5,6-TBA, and nitralin. Cytologia (Tokyo) 33(3/4): 545-554 (rec'd 1969).

190. Schiltz, P. 1969. Effects of picloram on the organogenesis of some Nicotianae. Seita Serv. Exploit Ind. Tabaes Allumettes Ann Dir Etud Quip Sect. 2 6: 189-198.

191. Schmutz, E.M. and D.E. Little. 1970. Effects of 2,4,5-T and picloram on broom snakeweed in Arizona. J. Range Manage. 23(5): 354-357.

192. Schneider, A.D., A.F. Wiese, O.R. Jones, and A.C. Mathers. (1971). Determining the fate of herbicides in the Ogallala Aquifer. Texas Agr. Exp. Sta. B-1112. 15 p.

193. Schrank, A.R. 1968. Growth and geotropic responses of Avena coleoptiles to 4-amino-3,5,6-trichloropicolinic acid. Physiol. Plant 21(2): 314-322.

194. Scifres, C. J. and R.W. Bovey. 1970. Differential responses of sorghum varieties to picloram. Agron. J. 62:775-777.

195. Scifres, C.J., R.W. Bovey, and M.G. Merkle. 1972. Variation in bioassay attributes as quantitative indicies of picloram in soils. Weed Res. (In Press).

196. Scifres, C.J., O.C. Burnside, and M.K. McCarty. 1969. Movement and persistence of picloram in pasture soils of Nebraska. Weed Sci. 17(4): 486-488.

197. Scifres, C.J., R.R. Hahn, and J. H. Brock. 1971. Chemical control as related to phenology of common broomweed. J. Range Manage 24:370-373.

198. Scifres, C.J., R.R. Hahn, J. Diaz-Colon, and M.G. Merkle. 1971. Picloram persistence in semiarid rangeland soils and water. Weed Science 19(4): 381-384.

199. Scifres, C.J., R.R. Hahn, and M.G. Merkle. 1971. Dissipation of picloram from vegetation of semi-arid rangelands. Weed Science 19(4): 329-331.

200. Scott, P.C. and R.O. Morris. 1970. Quantitative distribution and metabolism of auxin herbicides in roots. Plant Physiol. 46(5): 680-684.

201. Sharma, M.P., F.Y. Chang, and W. H. Vander Born. 1971. Penetration and translocation of picloram in Canada thistle. Weed Science 19(4): 349-355.

202. Sharma, M.P. and W.H. Vanden Born. 1970. Foliar penetration of picloram and 2,4-D in aspen and balsam poplar. Weed Science 18(1): 57-63.

203. Sharma, M.P. and W. H. Vanden Born. 1971. Effect of picloram on $^{14}CO_2$-fixation and translocation of ^{14}C assimilates in Canada thistle, soybean and corn. Can. J. Bot. 49(1): 69-74.

204. Shellhorn, S.J. and H. M. Hull. 1971. A carrier for some water-soluble herbicides. Weed Science 19(1): 102-106.

205. Shipman, R.D. 1971. Soil applied herbicides in the control of temperate zone grasses, broadleaf weeds and woody plants. Final Report. The Pennsylvania State University, University Park, Pennsylvania 16802. Contract No. DAAA13-69-C-0085 with Fort Detrick, Frederick, Maryland 21701. 140 p.

206. Sterrett, J.P. 1968. Response of oak and red maple to herbicides applied with an injector. Weed Sci. 16(2): 159-160.

207. Sterrett, J.P. 1969. Injection of red maple and hickory with picloram, 2,4,-D and 2,4,5-T. Down to Earth 25(2): 18-20.

208. Sterrett, J.P. 1969. Injection of hardwoods with dicamba, picloram, and 2,4-D. J. Forest. 67(11): 820-821.

209. Swanson, C.R. and J.R. Baur. 1969. Absorption and penetration of picloram in potato tuber discs. Weed Sci. 17(3): 311-314.

210. Swezey, A.W. and A. Montano. 1968. Chemical brush control and grass improvement in pastures in Central America. Down to Earth 24(1): 6-9.

211. The Dow Chemical Company. 1966. Determination of residues of tordon acid in wheat grain by gas chromatography. ACR 65.3R. 11 p. illus. Feb. 4.

212. The Dow Chemical Company. 1966. Determination of residues of tordon acid in wheat straw by gas chromatography. ACR 66.5. 12p. illus. May 6.

213. The Dow Chemical Company. 1967. Determination of residues of tordon acid in bovine tissues by gas chromatography. ACR 67.2. 17 p illus. June 21.

214. The Dow Chemical Company. 1967. Determination of residues of tordon acid in milk by gas chromatograpny. ACR 67.3. 11 p. illus. June 22.

215. The Dow Chemical Company. 1968. Determination of 4-amino-3,5,6-trichloropicolinic acid in water. ACR 68.14. 16p. illus. Sept. 26.

216. The Dow Chemical Company. 1968. Residue determination method--Gas chromatographic determination of residues of 4-amino-3,5,6-trichloropicolinic acid in soil treated with tordon herbicide. ACR 68.7. 13 p. illus. June 7.

217. Trichell, D.W., H.L. Morton, and M.G. Merkel. 1968. Loss of herbicides in runoff water. Weed Sci. 16(4): 447-449.

218 Tsay, Ruey-chyong, and F.M. Ashton. 1971. Effect of several herbicides on dipeptidase activity of squash cotyledons. Weed Science 19(6): 682-684.

219. Tschirley, F.H. 1967. Problems in woody plant control evaluation in the tropics. Weeds 15(3): 233-237.

220. Tu, C.M. and W.B. Bollen. 1969. Effect of tordon herbicides on microbial activities in three Willamette Valley soils. Down to Earth 25(2): 15-17.

221. Tueller, P. T. and R.A. Evans. 1969. Control of green rabbitbrush and big sagebrush with 2,4-D and picloram. Weed Sci. 17(2): 233-235.

222. Upchurch, R.P., H.D. Coble, and J.A. Keaton. 1969. Rainfall effects following herbicidal treatment of woody plants. Weed Sci. 17(1): 94-98.

223. Valentine, K.A. 1970. Creosotebush control with phenoxy herbicides, picloram, and fuel oil in Southern New Mexico. N. Mex. Agr. Exp. Sta. Bull 554: 3-12.

224. Vanden Born, W.H. 1965. The effect of dicamba and picloram on quackgrass, bromegrass, and Kentucky bluegrass. Weeds 13(4): 309-312.

225. Van Schreven, D.A., D. J. Linderbergh, and A. Koridon. 1970. Effect of several herbicides on bacteria populations and activity and the persistence of these herbicides in soil. Plant Soil 33(3): 513-532.

226. Vernie, F., J. Lhoste, and A. Casanova. 1966. (France). Trial results with picloram for weed control in winter wheat. Weed Res. 6(4); 322-331.

227. Victoria, J., A. Sanchez, and R. Barriga. 1970. Eradication of coconut palms affected with red ring disease (Rhadinaphelenchus cocophilus Cobb 1919 Goodey 1960, Nematoda: Aphelenchoididae), by the use of chemical substances. Rev inst. Colomb. Agropecuar 5(3): 185-197.

228. Warden, R.L. 1964. Tordon...for the control of field bindweed and Canada thistle in the North Central United States. Down to Earth 20(2): 6-10.

Watson, A.J. and M.G. Wiltse. 1963. Tordon...for brush control on utility rights-of-way in the Eastern United States. Down to Earth 19(1): 11-14.

229. Wax, L.M. L.A. Knuth, and F.W. Slife. 1969. Response of soybeans to 2,4-D, dicamba and picloram. Weed Sci. 19(3): 388-393.

230. Weed Sci. Soc. Amer. 1967. Herbicides Handbook. W.F. Humphrey Press, Inc., Geneva, N.Y. 293 p.

231. Whipple, S.D. and K.P. Moeck. 1968. Potential uses of Tordon 10K pellets in forest management. Down to Earth 24(1): 13-17.

232. Whiting, F.L. and J. M. Lyons. 1971. A versatile tractor-mounted research spray system. Weed Science 19(6)): 743-745.

233. Wicks, G.A., C.R. Fenster, and O.C. Burnside. 1969. Selective control of plains prickly pear in rangeland with herbicides. Weed Sci. 17(4): 408-411.

234. Wiese, A.F., J. Gibson, and D. Lavake. 1967. Controlling large field bindweed infestations with repeated applications of tordon. Down to Earth 23(2): 2, 37-39.

235. Wilson, J.H. 1967. A bio-assay of Tordon solutions. Rhodesia Zambia Malawi J. Agr. Res. 5(3): 307-308.

236. Wilson, J.H. 1967. The effects of basal injections of Tordon on some central African indigenous trees. Rhodesia Zambia Malawi J. Agr. Res. 5(3): 301-303.

237. Woolson, E.A. and C.I. Harris. 1967. Methylation of herbicides for gas chromatographic determination. Weeds 15(2): 168-170.

238. Young, N.D. 1968. Tordon for eucalyptus control--a tool for land development in Australia. Down to Earth 24(3): 2-6.

239. Youngson, C.R., C.A. I. Goring, R.W. Meikle, H.H. Scott, and J. D. Griffith. 1967. Factors influencing the decomposition of tordon herbicide in soils. Down to Earth 23(2): 3-11.

240. Goring, C.A.I. and J.W. Hamaker. 1971. A Review...The degradation and movement of picloram in soil and water. Down to Earth 27(1): 12-15.

241. Hamaker, J.W., C.R. Youngson, and C.A.I. Goring. 1967. Prediction of the persistence and activity of Tordon herbicide in soils under field conditions. Down to Earth 23(2): 30-36.

REPORT

ON

BACKGROUND INFORMATION

FOR

THE PHENOXY HERBICIDES

2,4-D - 2,4,5-T - 2,4,5-TP

COMMITTEE MEMBERS:

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TABLE OF CONTENTS

TABLE OF CONTENTS

(Continued)

FOREWORD

The task of gathering and assembling background information on the three phenoxy herbicides (2,4-D, 2,4,5-T and 2,4,5-TP) becomes rather formidable.

This group of herbicides have been successfully used over a wide spectrum since the late 1940's. There is probably more known and more been written about this group of compounds than any other group.

For these reasons, the Committee established some ground rules:

1. Only the formulations which are recommended for range and forestry use were considered. There are hundreds of formulations which could be listed but would serve no useful purpose in the work of the Forest Service.

2. The three phenoxys 2,4-D, 2,4,5-T and 2,4,5-TP are included in one report. The three compounds are so similar in many respects and most of the literature refers to two and sometimes all three in regard to environmental impacts and residues. Much duplication was avoided in the approach the committee followed.

When necessary, detailed information is included on each herbicide.

PHENOXY HERBICIDES

Section I

I. General Information

The phenoxy herbicides 2,4-D 2,4-dichlorophenoxyacetic acid; 2,4,5-T, 2,4,5-trichlorophenoxyacetic acid; silvex, 2-(2,4,5 trichlorophenoxy) propionic acid are registered by the Environmental Protection Agency for use on forest and range land and on utility right-of-ways. 2,4-D is most widely used to control herbaceous weeds on agriculture crop land, and is registered for use on orchards, vegetable fields, berries, vineyards, grain and hay crops, fallow land and pastures. 2,4-D along with 2,4,5-T and Silvex, is also a valuable herbicide for controlling many woody plants on forest and rangeland. The label details all the registered uses. If a use is not on the label, it is not registered for that use.

At the present time the pure acid of the phenoxys are almost never used as herbicides. In the past they found limited use when formulated as an emulsifiable acid. The earliest widespread use of these chemicals was an inorganic salts of the acids. These formulations proved to be of limited value and have generally gone out of use, although some sodium salt of 2,4-D is still used in general agriculture on certain crops.

The water soluble and soil soluble amine sales account for less than 10 percent of total use of Phenoxy herbicides for forestry and range purposes. Amines are less volatile than the ester forms of these herbicides and are used where the vapors of the esters could cause damage to nearby susceptible species. Water-soluble amines are usually used for cut surface or injection into individual stems. This treatment is highly selective and safe, but is expensive in time and labor. However, the method is justified where values are high and there are relatively few (not more than 200) stems per acre. If the number of stems is high, an oil-soluble ester can be used in oil and is applied as a basal spray without bark incision. This method is less effective and usually gives unsatisfactory control except upon highly susceptible thin-barked plants especially during the growing season. The oil-soluble esters are usually more satisfactorily used as basal sprays. Oil-soluble amines can be used as foliage sprays upon susceptible species where the volatile vapors of even low volatile esters of 2,4-D may be a hazard to nearby susceptible crops or plants.

Esters of the phenoxy herbicides may be either high volatile or low volatile depending upon the length of the carbon chain of the alcohol used to formulate the herbicide. Low volatile esters are usually used in foliage sprays and provide satisfactory results on a wide spectrum of species. High volatile esters are not recommended for use on forest and rangeland because volatile vapors may damage nearby non-target species.

At the present time, low volatile esters are used on at least 80 to 90 percent of all forest and range improvement spray projects.

Formulations

The following list contains formulations which are recommended for use on range and timber areas. The label of any chemical container should be studied carefully, and the information relied upon and adhered to. This information is derived from much research and is part of the labeling and registration process for herbicides which is carefully regulated by federal and state agencies. Some typical forms of phenoxy herbicides are:

A. Esters

1. Low volatile
    a. Propylene glycol butyl ether
    b. Butoxy ethauol
    c. Isooctyl

2. High volatile
    a. Isopropyl
    b. Butyl, n-Butly and isobutyl
    c. Ethyl

B. Amines

1. Water soluble
    a. Dimethyl amine
    b. Triethanol amine
    c. Triisoproponol amine

2. Oil soluble

   a. Dodecyl amine

   b. Tetradecyl amine

   c. N-oleyl-1,3,-propylene diamine

C. Parent Acid

D. Inorganic salts

1. Sodium

2. Potassium

3. Lithium

4. Ammonium

Table 1 Trade names, chemical formulations, and manufacturers of products containing two or more pounds of acid equivalent (a.e.) per gallon of product.

2,4,5-T (Low Volatile)

Manufacturer	Trade Name	Formulation	Strength
Dow	Esteron 245	Propylene glycol butyl ether esters	4 lbs. a.e./gal.
Dow	Esteron 245 conc.	Propylene glycol butyl ether esters	6
Thompson-Hayward	Ded-weed LV 6	Isooctyl ester	4
Thompson-Hayward	Ded-weed LV 9	Isooctyl ester	6
Amchem	Weedone, 2,4,5-T	Butoxy ethanol ester	4
Amchem	Trinoxol	Butoxy ethanol ester	4
Amchem	Trinoxol super 6	Butoxy ethanol ester	6
Rhodia	Chipman, product 138	Isooctyl ester	4
Diamond Shamrock	Brush/killer Lo-Vol 4T	Isooctyl ester	4
Diamond Shamrock	Brush/killer Lo-Vol 6T	Isooctyl ester	6
Dow	Veon 245	Triethylamine salt	4
Thompson-Hayward	Ded-weed amine T	Triethylamine salt	4
Amchem	Weedar	Triethylamine salt	4
Diamond Shamrock	Croprider amine 4T	Triethylamine salt	4
Amchem	Emulsamine 2,4,5-T	Alkyl(C14) amine salt	3
Rhodia	Visko=Rhap. Prod. 713	N,N-Dimethyl oleyl linoleyl amine salt	3
Diamond Shamrock	Dacamine 4T	N-oleyl-1,3 propylene diamine salt	4
Dow	Esteron brush killer	Propylene glycol butyl ether esters	2 lbs. of each
Dow	Verton C.E.	Propylene glycol butyl ether esters	2 lbs. of each
Hayward-Thompson	Ded-weed LV 33	Isooctyl esters	2
Amchem	Dinoxol	Butoxyethanol esters	2
Rhodia	Chipman product 112	Isooctyl esters	2
Diamond Shamrock	Brush killer Lo-Vol 2D/2T	Isooctyl esters	2
Dow	Veon brush killer	Tri and Di ethylamine salts	2
Amchem	Weedar	Tri and Di ethylamine salts	2
Diamond Shamrock	Brush killer Dimethyl amine salts		2
Amchem	Emulsamine brush killer	Alkyl (C12) and alkyl (C14) amine salts	1.5 lbs. each
Diamond Shamrock	Brush kill. dacamine 2D/2T	N oleyl 1,3 propylenediamine salts	2

Table II. Trade names, chemical formulations and manufacturers of products containing two or more pounds acid equivalent (a.e.) per gallon of product.

2,4,5-TP (Silvex)

MANUFACTURER	TRADE NAME	FORMULATION	STRENGTH
Diamond Shamrock	Crop Rider	Isooctyl ester	4 lbs. a.e./Gal.
Dow	Kuron	Propylene glycol butyl ester	4 lbs. a.e./Gal
Hercules	Silvi Rhap	2 Ethylhexyl ester	4 lbs. a.e./Gal.
Amchem	Weedone	Butoxyethanol ester	4 lbs. a.e./Gal.
Rhodia	Chipman	Low volatile ester	4 lbs. a.e./Gal.
Miller	Silvicide	Potassium Salt	4 lbs. a.e./Gal.

Table III. Trade names, chemical formulations and manufacture of products containing two or more pounds acid equivalent (a.e.) per gallon of product.

2,4,D (Low Volatile)

MANUFACTURER	TRADE NAME	FORMULATION	STRENGTH
Diamond	Crop Rider	Isooctyl ester	4-6 lbs. a.e./Gal.
Monsanto	Field Clean	Isooctyl ester	6 lbs. a.e./Gal.
Chevron Chem.	Ortho	Isooctyl ester	4 lbs. a.e./Gal.
Stauffer	Stauffer 2,4-D	Isooctyl ester	4 lbs. a.e./Gal.
Rhodia	Chipman	Isooctyl ester	4-6 lbs. a.e./Gal.
Dow	Esteron 99	Propylene glycol butyl ester	4-6 lbs. a.e./Gal.
Hercules	Weed Rhap	Ethylhexyl ester	4 lbs. a.e./Gal.
Monsanto	Amine Weed Killer	Dimethyl amine	4 lbs. a.e./Gal.
Miller Chem.	Hormotox	Dimethyl amine	4 lbs. a.e./Gal.
Diamond	Crop Rider	Dimethyl amine	4-6 lbs. a.e./Gal.
Chipman	Chipman Amine No. 4-- Amine No. 6	Dimethyl amine " "	4-6 lbs. a.e./Gal.
Chevron Chem.	Ortho	Dimethyl amine	4 lbs. a.e./Gal.
Stauffer	--	Dimethyl amine	4 lbs. a.e./Gal.
Diamond	Dacamine	N-oleyl-1,3-propylen-diamine	4-6 lbs. a.e./Gal.

Dilutions of Formulation for Use

Hormone type herbicides are highly active in a biological sense, therefore very small amounts are required to obtain desired results. For this reason they are always diluted with a carrier to obtain the desired distribution over the sprayed area and coverage of the spray droplets over the leaf and stem surfaces. An equal volume of large droplets cannot be substituted for the same volume composed of a large number of small droplets. For example, 100 small droplets on a leaf may be equal to 1/10th the total volume of one large droplet. The combined effect of the small droplets, although containing only 1/10th as much spray, may be many times more than the effect of the one large droplet. About 75 droplets per square inch, regardless of their size, are required for a satisfactory effect of phenoxy hormone type herbicides (Behrens 1957). Droplets should not be reduced in size too much however, since the probability of drift increases with decreasing droplet size. About 200-300 microns volume mean diameter (VMD) is about the smallest droplet that can be used without excessive drift hazard (Akesson, Wilce, and Yates 1971). Their recommendation is 450 micron VMD for aircraft spraying. Total gallonage per acre must be increased to maintain the necessary number of droplets per square inch if droplet sizes exceed 800 microns VMD.

Individual stem treatments can be made with the undiluted concentrate, however, they are usually diluted with water or diesel oil to reduce the amount of chemical used. Carriers or dilutents are usually used

for foliar and other broadcast applications. The most common carriers for foliar applications are diesel oil, diesel oil-water emulsions, and water. Manufacturer's labels list recommended carriers and specific mixing directions and should always be read carefully and followed.

Water is used with water-soluble amines and with emulsifiable acids and esters to form emulsions. Water carriers are usually used early in the season before leaf cuticles thicken. Older plants with thin cuticles may also be sprayed with water dilutents.

Oil-water emulsions (1/2 to 1 gpa of oil) are usually considered to be better than a straight water carrier, and are as effective and are usually cheaper than a straight oil carrier. If large amounts of clean water have to be transported long distances, they sometimes can become more expensive than lesser amounts of diesel oil. Lower volumes of sprays using diesel oil dilutents can often be used with equal satisfaction as higher volumes of water-oil emulsion sprays. Oil dilutents have lower surface tensions than water and therefore dispurse and spread better which tends to make them more effective carriers than water. Caution should be used when exceeding 5 gallons of diesel oil per acre, for the oil itself is somewhat phytotoxic and may kill and dessicate tissue of the leaves, thus cutting down or eliminating herbicide absorption.

Rate and Method of Application

There are two basic methods of herbicide application. (1) Broadcast and (2) individual plant treatment. These may be further divided as follows:

A. Broadcast spraying

1. Aerial spraying
    a. Helicopter
    b. Fixed wing aircraft

2. Ground rig spraying
    a. Boom spraying
    b. Broadjet
    c. Mist blower
    d. High volume-hand gun

B. Individual plant treatment

1. Foliage spraying
    a. Hand gun
        (1) Power sprayer (may be high volume or low volume per acre depending upon plant numbers and volume needed for adequate coverage)

        (2) Backpack sprayer (low volume per acre, few small plants)

    b. Backpack mist blower - may be broadcast spraying if plant numbers are high.

2. Stem treatments

   a. Injections

   b. Frills

   c. Stem sprays

3. Stump treatments.

Broadcast spraying is aimed at covering the foliage of all the plants on the target area with an adequate amount of herbicide to bring about the desired results. Broadcast spraying is usually done at rather low volumes of 3 to 20 gallons of carrier per acre. High volume ground sprays, 100 to 200 gpa with a truck-mounted power sprayer and hand gun are usually restricted to roadsides or to other rights-of-way and industrial sites. From 1/2 to 4 pounds a.e. of herbicide per acre are usually applied in broadcast sprays. The most common rate is about 2 pounds per acre. High volume sprays usually contain about four pounds a.e. herbicide per 100 gallons of spray solution.

When doing individual plant foliage spraying, spray solutions are usually mixed the same as for high volume spraying at two to six pounds per acre per hundred gallons (AHG). The foliage of each plant is sprayed until wet to runoff. When there are high numbers of plants per acre, these treatments may exceed 200 or more gpa. For this reason, if plant numbers to be sprayed exceed 100 to 150 per acre, it is usually cheaper to broadcast spray. Of course, if there are certain susceptible plants on the area which must be saved, broadcast spraying cannot be used.

Stem treatments are widely used to remove undesirable plants from a stand. Again, this is a highly selective but costly method if large numbers of plants are to be treated. The following are the most commonly used mixes for these treatments.

Pounds of Herbicide (a.e./100 gal.)

	Range	Most Common
Injection	10 - 30	20
Basal or stump spray	16 - 20	16
Frill 1/	4 - 20	8

1/ Sometimes the pure amine formulation is used without dilution. Stump treatments may be either sprayed to wet the freshly cut top and sides or the chemical can be painted on liberally with a paint brush. The spray mix is usually about the same as used for a basal stem spray.

Inverted or thickened emulsions can be used to create larger spray droplets and to help avoid drift. There are several thickening agents which can be added to spray solutions. Also, small amounts of water in large amounts of oil will create an inverted emulsion. These thickened sprays have a larger average droplet size which reduces, but seldom eliminates, spray drift hazard. Thickened or invert sprays still have a large range of droplet sizes. The smaller

droplets are subject to drift and the large drops may not give adequate coverage of droplets per square inch. When using thickened sprays, it may be necessary to increase the gallons per acre to maintain the coverage necessary for the spray to be effective. In general, the inverted or thickened sprays have not given as good results as normal spray emulsions.

Thickening agents and inverts offer considerable promise for safe application of herbicides. Each of the materials have certain advantages and limitations. Successful use requires a knowledge of its specific characteristics. Drift control is accomplished by a reduction in percentage of small drift susceptible droplets in the spray. Nozzle tip design and orientation, solution, viscosity, air speed and sprayer pressure appear to be important factors factors in determining the success of thickened sprays.

Regulating droplet breakup holds promise in controlling drift.

The Micro Foil Boom used with a helicopter, provides a spray having a minimum of large and small droplets. Good coverage is obtained with moderate volumes of carrier. Special care is required to maintain the system free of foreign matter which would plug the small orifices. The system has not been evaluated with fixed-wing aircraft. Further evaluation is necessary to determine the potential of this equipment for controlling drift of herbicide sprays applied in rangeland brush and weed

control. Additional research is needed to develop methods of regulating spray breakup with fixed-wing aircraft application.

Tolerances and Safety

Tolerances have been established for 2,4,-D in food and feeds. It is expected that these petitions will be sufficient for fruits and vegetable crops. New data on residue standards are being developed for grass with respect to pasture and range usage.

Amendments to the petitions for tolerances for 2,4-D, Silvex, and 2,4,5-T have been submitted to the appropriate agency for inclusion in uses for pasture and rangeland tolerances. At present, tolerances for 2,4-D residues in feed and grains is 0.5 ppm; for forages it is 20 ppm. Some official tolerances have not been established yet, and it should be understood that the tolerances listed do not necessarily represent the potential hazard, but rather represent the amount expected with good operational practices. In no case can the tolerance be greater than the established safety threshhold, and in most instances it is considerably lower. Where more than one herbicide is involved, such as in brushkiller formulations, the total residue for all herbicides cannot exceed the lowest established tolerance threshhold for any one of the herbicides in the mixture. In brushkiller for example, the established tolerance for either 2,4-D or 2,4,5-T whichever is the lowest for that crop would apply.

Hormone type herbicides degrade quite rapidly and will under most conditions be well within the tolerance levels if time intervals, dosages, and other directions specified on the label are followed carefully. 2,4-D, 2,4,5-T and Silvex have a low direct toxicity to man. However, some persons may be allergic to the chemicals or to the oil used in the herbicidal mixtures, so skin contact should be avoided. Gloves, goggles, and protective clothing should be available and when there is spray mist in the air, a respirator is also a desirable piece of safety equipment. If any nausea or skin rash is observed, directions in the Forest Service Health and Safety code should be followed. If a doctor is consulted, information about the chemical and mixtures being used should be made available to him.

REFERENCES FOR I - GENERAL INFORMATION

1. Akesson, Norman B., Stephen E. Wilce, and Wesley E. Yates. 1971. Confining aerial applications to treated fields -- a realistic goal. Agrichemical Age. December 1971, p. 11-14.

2. Akesson, Norman B., Wesley E. Yates, and Stephen E. Wilce. 1970. Controlling spray atomization. Agrichemical Age. December 1970, p. 10-17.

3. Agricultural Extension Service. 1967. Proceedings - aerial applicator's short courses. University of California.

4. Bailey, J. Blair and John E. Swift. 1968. Pesticide information and safety manual. Univ. of Calif. Agric. Extension Service. Agricultural Experiment Station.

5. Behrens, Richard. 1957. Influence of Various Compounds on the Effectiveness of 2,4,5-T Sprays. Weeds, 5:183-186.

6. Bentley, Jay R. and Kenneth M. Estes. 1965. Use of herbicides on timber plantations. California Region and Pacific Southwest Forest and Range Experiment Sta., San Francisco, California

7. Bentley, Jay R. 1967. Conversion of chaparral areas to grassland -- techniques used in California. Agriculture Handbook No. 328. U. S. Dept. of Agriculture, Forest Service.

8. Butler, B. J., N. B. Akesson, and W. E. Yates. 1969. Use of spray adjuvants to reduce drift - transactions of the ASAE, Vol. 12(2): 182-186.

9. Dunham, R. S. 1965. Herbicide manual for noncropland weeds. Agriculture Handbook No. 269, Agricultural Research Service.

10. Hayes, Wayland J., Jr. 1963. Clinical handbook on economic poisons. U. S. Dept. of Health, Education, and Welfare, Public Health Service, Communicable Disease Center, Atlanta, Georgia.

11. Hoffman, Garlyn O. and Robert H. Haas. Controlling Drift of Herbicides. Fact sheet, L848. Texas A&M University, College Station, Texas.

12. Kaupke, C. R. and W. E. Yates. 1966. Physical properties and drift characteristics of viscosity-modified agricultural sprays. Transactions of the ASAE, Vol. 9(6):797-799; 802.

13. Leonard, O. A. and W. A. Harvey. 1965. Chemical control of woody plants. California Agricultural Experiment Sta. Bulletin 812.

14. Montgomery, Marvin L. and Logan A. Norris. 1970. A preliminary evaluation of the hazards of 2,4,5-T in the forest environment U.S.D.A. Forest Service, Pacific Northwest Forest and Range Expt. Sta. Research Note PNW-116.

15. Norris, Logan A. 1971. Chemical Brush control: assessing the hazard. Jour. Forestry, October 1971 p. 715-720.

16. Romancier, Robert M. 1965. 2,4-D, 2,4,5-T, and related chemicals for woody plant control in the southeastern United States. Georgia Forest Research Council, Macon, Georgia, Report No. 16.

17. Weed Society of America. 1967. Herbicide Handbook of the Weed Society of America. First Edition. W. F. Humphrey Press Inc., Geneva, New York.

18. Whitworth, J. W. and W. P. Anderson. 1969. Accurately predicting a herbicide's potential. Weed Sci. Vol. 17(3):290-293.

Section IL

ACUTE TOXICITY OF 2,4-D

Formulation	Organism	Dose	Effect	Reference
Butoxyethanol ester	Oyster	3.75 ppm(96hrs)	50% decrease in shell growth	Butler (1965)
Butoxyethanol ester	Shrimp	1 ppm(48 hrs)	No effect	Butler (1965)
Butoxyethanol ester	Fish (Salt water)	5 ppm	48 hr TLm	Butler (1965)
Butoxyethanol ester	Phytoplankton	1 ppm	16% decrease in CO_2 fixation	Butler (1965)
Dimethylamine	Oyster	2 ppm (96 hrs)	No effect on shell growth	Butler (1965)
Dimethylamine	Shrimp	2 ppm (48 hrs)	10% mortality or paralysis	Butler (1965)
Dimethylamine	Fish(salt water)	15 ppm (48 hrs)	No effect	Butler (1965)
Dimethylamine	Phytoplankton	1 ppm (4 hrs)	No effect on CO_2 fixation	Butler (1965)
Ethylhexyl ester	Oyster	5 ppm (96 hrs)	38% decrease in shell growth	Butler (1965)
Ehtylhexyl ester	Shrimp	2 ppm (48 hrs)	10% mortality or paralysis	Butler (1965)
Ethylhexyl ester	Fish(salt water	10 ppm(48 hrs)	No effect	Butler (1965)
Ethylhexyl ester	Phytoplankton	1 ppm (4 hrs)	49% decrease in CO_2 fixation	Butler (1965)
PGBE 1/ester	Oyster	1 ppm (96 hrs)	39% decrease in shell growth	Butler (1965)
PGBE 1/ester	Shrimp	1 ppm (48 hrs)	No effect	Butler (1965)
PGBE 1/ester	Fish(salt water)	4.5 ppm	48 hr TLm	Butler (1965)
PGBE 1/ester	Phytoplankton	1 ppm (4 hrs)	44% decrease in CO_2 fixation	Butler (1965)

1/ PGBE is propylene glycol butyl ether

Formulation	Organism	Dose	Effect	Reference
Alkanolamine	Chick	380-765 mg/kg	LD_{50}	Rowe, et al.(1954
Isopropyl ester	Rat	700 mg/kg	LD_{50}	Rowe, et al.(1954
Isopropyl ester	Chicks	1420 mg/kg	LD_{50}	Rowe, et al.(1954
Isopropyl ester	Guinea pig	550 mg/kg	LD_{50}	Rowe, et al.(1954
Butyl ester	Rat	620 mg/kg	LD_{50}	Rowe, et al.(1954
Butyl ester	Guinea pig	848 mg/kg	LD_{50}	Rowe, et al.(1954
Butyl ester	Chicks	2000 mg/kg	LD_{50}	Rowe, et al.(1954
PGBE	Rat	570 mg/kg	LD_{50}	Rowe, et al.(1954
Acid	Dog	100 mg/kg	LD_{50}	Rowe, et al.(1954
Acid	Chick	541 mg/kg	LD_{50}	Rowe, et al.(1954
Dimethylamine	Bluegill	166 ppm	48 hr TLm	Lawrence (1966)
Alkanolamine	Bluegill	435 ppm	48 hr TLm	Lawrence (1966)
Isooctyl ester	Bluegill	9 ppm	48 hr TLm	Lawrence (1966)
Butyl ester	Bluegill	1 ppm	48 hr TLm	Lawrence (1966)
Isopropyl ester	Bluegill	1 ppm	48 hr TLm	Lawrence (1966)
PGBE	Bluegill	3 ppm	48 hr TLm	Hughes&Davis (196
Triethanolamine	Swine	50 mg/kg	No effect	Bjorklund & Erne (1966)
Triethanolamine	Swine	500 mg/kg	Lethal	Bjorklund & Erne (1966)
Butyl ester	Swine	100 mg/kg	No effect	Bjorklund & Erne (1966)
Triethanolamine	Chicken	300 mg/kg	No effect	Bjorklund & Erne (1966)
Butyl ester	Rat	620 mg/kg	LD_{50}	Edson et al.(1964
Isopropyl ester	Rat	700 mg/kg	LD_{50}	Hayes, (1963)
Unspecified amine	Mallard duck	2000 mg/kg	LD_{50}	Tucker & Crabtree (1970)
Acid	Pheasant	472 mg/kg	LD_{50}	Tucker & Crabtree (1970)
Acid	Mule deer	400-800 mg/kg	LD_{50}	Tucker & Crabtree (1970)

*Footnote: dermal 300-1500 mg/kg various formulations

ACUTE TOXICITY OF 2,4,5-T

Oral Formulation	Organism	Dose	Effect	Reference
Acid	Rat	500 mg/kg	LD 50	Rowe & Hymas (1954)
Isopropyl ester	Mice	551 mg/kg	LD 50	Rowe & Hymas (1954)
Butyl ester	Mice	940 mg/kg	LD 50	Rowe & Hymas (1954)
Amyl ester	Rat	750 mg/kg	LD 50	Rowe & Hymas (1954)
Isooctyl esters (From 3 manufacturers)	Bluegill	10-31 ppm	48 TLm	Hughes & Davis (1963)
PGBE ester	Bluegill	17 ppm	48 TLm	Hughes & Davis (1963)
Butoxyethanol ester	Bluegill	1.4 ppm	48 TLm	Hughes & Davis (1963)
Triethanolamine	Swine	100 mg/kg	Locomotory disturbance	Bjorklund & Erne (1966)
PGBE ester	Oyster	0.14(96 hrs)	50% decrease in shell growth	Butler (1965)
PGBE ester	Shrimp	1 ppm(48 hrs)	20% mortality or paralysis	Butler (1965)
PGBE ester	Fish(salt water)	0.32 ppm	48 hr TLm	Butler (1965)
PGBE ester	Phytoplankton	1 ppm(4 hrs)	89% decrease in CO_2 fixation	Butler (1965)
Veon 2,4,5	Oyster	1 ppm(96 hrs)	No effect	Butler (1965)
	Shrimp	1 ppm(48 hrs)	No effect	Butler (1965)
	Fish(Salt water)	1 ppm(48 hrs)	No effect	Butler (1965)
	Phytoplankton	1 ppm(4 hrs)	No effect on CO_2 fixation	Butler (1965)

ACUTE TOXICITY OF 2,4,5-TP

Oral Formulation	Organism	Dose	Effect	Reference
PGBE ester	Rat	650 mg/kg	LD_{50}	Bailey & Swift (1968)
PGBE ester	Rat	1070 mg/kg	LD_{50}	Mullison(1966)
PGBE ester	Guinea pig	850 mg/kg	LD_{50}	Mullison(1966)
PGBE ester	Rabbit	850 mg/kg	LD_{50}	Mullison(1966)
PGBE ester	Mouse	2140 mg/kg	LD_{50}	Mullison(1966)
PGBE ester	Chicken	2000 mg/kg	LD_{50}	Mullison(1966)
Acid	Mallard Duck	500 mg/kg	Minor symptoms	Tucker & Crabtree (1970)
Acid	Mallard Duck	2000 mg/kg	LD_{50}	Tucker & Crabtree (1970)
Isooctyl ester	Bluegill	5 ppm	48 hr TLm	Hughes&Davis (1966)
PGBE ester	Bluegill	25 ppm	48 hr TLm	Hughes&Davis (1966)
Butoxyethanol ester	Bluegill	2 ppm	48 hr TLm	Hughes&Davis (1966)
Triethylamine	Bluegill	20 ppm	48 hr TLm	Hughes&Davis (1966)
PGBE ester	Oyster	1 ppm for 96 hrs	23% decrease in shell growth	Butler (1965)
PGBE ester	Shrimp	0.24 ppm(48 hrs)	50% mortality or paralysis	Butler (1965)
PGBE ester	Fish(Salt water)	0.36 ppm	48 hr TLm	Butler (1965)
PGBE ester	Phytoplankton	1 ppm (4 hrs)	94% decrease CO_2 fixation	Butler (1965)
Acid	Rat	650 mg/kg	LD_{50}	Rowe & Hymas (1954)
Butyl ester	Rat	600 mg/kg	LD_{50}	Rowe & Hymas (1954)
PGBE ester	Rat	621 mg/kg	LD_{50}	Rowe & Hymas (1954)
PGBE ester	Guinea pig	1250 mg/kg	LD_{50}	Rowe & Hymas (1954)
PGBE ester	Rabbit	819 mg/kg	LD_{50}	Rowe & Hymas (1954)
PGBE ester	Chick	1190 mg/kg	LD_{50}	Rowe & Hymas (1954)

CHRONIC TOXICITY OF 2,4-D

Formulation	Organism	Dose	Duration	Effect	Reference
Triethanolamine	Swine	50/mg/kg/day	3 doses	None	Bjorklund & Erne (1966)
Triethanolamine	Swine	50/mg/kg/day	8-10 doses	Minor transient effects	Bjorklund & Erne (1966)
Butyl ester	Swine	50/mg/kg/day	<5 doses	None	Bjorklund & Erne (1966)
Triethanolamine	Swine	500 ppm in feed.	1 month	Some locomotory disturbance, depressed growth rate, no gross pathology	Bjorklund & Erne (1966)
Triethanolamine	Rats	1000 ppm in water	10 mos.	Depressed growth rate, no gross pathology	Bjorklund & Erne (1966)
Triethanolamine	Chicken	1000 ppm in water	Daily from hatching through first 2 mos. of egg production	Egg size normal, production reduced 30%	Bjorklund & Erne (1966
Alkanolamine	Sheep	100/mg/kg/day	481 days	No effect	Palmer & Radeleff (1964)
Alkanolamine	Cattle	50/mg/kg/day	112 days	No effect	Palmer & Radeleff (1964)
PGBE ester	Sheep	100/mg/kg/day	481 days	No effect	Palmer & Radeleff (1964)
Ethylhexyl ester	Cattle	250/mg/kg/day	14 days	Ill in 3 days, survive & recover from 9 doses. 14 doses lethal.	Hunt, et. al. (1970)
Ethylhexyl ester	Sheep	250/mg/kg/day	17 days	Ill in 3 days 17 doses lethal	Hunt, et. al. (1970)
Ethylhexyl ester	Sheep & Cattle	100/mg/kg/day	10 days	None to minor effects	Hunt, et. al. (1970)
Not specified	Dog	500 ppm in feed	2 years	None	House et. al. (1967)

Formulation	Organism	Dose	Duration	Effect	Reference
Not specified	Rat	1250 ppm in feed	2 years	No effects on growth, survival hermatology or tumor incidence.	House, et. al (1967)
Not specified	Rat	500 ppm in feed	2 years	No effects in reproduction studies.	House, et. al (1967)
Alkanolamine	Chicken	100 mg/kg/day	10 days	No effect on weight gain	Palmer & Radeleff (196
PGBE ester	Chicken	50 mg/kg/day	10 days	No effect on weight gain	Palmer & Radeleff (196
PGBE ester	Cattle	100 mg/kg/day	10 days	No effect	Palmer & Radeleff (196
Acid	Mule deer	80 and 240 mg/kg/day	30 days	Minor symptoms no weight loss	Tucker and Crabtree(1970

CHRONIC TOXICITY OF 2,4,5-T

Formulation	Organism	Dose	Duration	Effect	Reference
Not specified	Dog	10 mg/kg/day	5 days per wk. for 90 days	Minor weight loss, no other effects.	Drill & Hiratzka (1953)
Not specified	Dog	20 mg/kg/day	5 days per wk. for 90 days	Lethal between 11 and 75 days	Drill & Hiratzka (1953)
PGBE ester	Cattle	100 mg/kg/day	10 days	None	Palmer & Radeleff(1969)
PGBE ester	Sheep	50 mg/kg/day	10 days	None	Palmer & Radeleff(1969)
PGBE ester	Sheep	100 mg/kg/day	369 days	(dosed by capsule) Ill at 367 doses, lethal at 369.	Palmer & Radeleff(1969)
PGBE ester	Chicken	100 mg/kg/day	10 days	No effect on weight gain	Palmer & Radeleff(1969)
Triethylamine	Sheep	100 mg/kg/day	481 days	No effect	Palmer & Radeleff(1964)
Not specified	Mice	21 mg/kg/day 600 ppm in diet.	4 weeks 18 months	No mortality	Inues, et. al. (1969)

CHRONIC TOXICITY OF 2,4,5-TP

Formulation	Organism	Dose	Duration	Effect	Reference
Butoxyethanol ester	Quail	5000 ppm in feed	10 days	LD_{50} 9350 mg/kg	House, et. al. (1967)
Butoxyethanol ester	Mallard Duck	2500 ppm in feed	13 days	LD_{50} 33700 mg/kg	House, et. al. (1967)
Butoxyethanol ester	Pheasants	5000 ppm in feed	<100 days	LD_{50} 9240 mg/kg	House, et. al. (1967)
PGBE ester	Rat	30 mg/kg	90 days	No effect	House, et. al. (1967)
Not specified	Rat	100 ppm feed	2 years	No effect	House, et. al. (1967)
Not specified	Dog	190 ppm feed	2 years	No effect	House, et. al. (1967)
PGBE ester	Sheep	100 mg/kg	11 doses	Lethal	Palmer & Radeleff(1964)
PGBE ester	Cow	50 mg/kg	73	No effect	Palmer & Radeleff(1964)
PGBE ester	Cow	100 mg/kg	29	Lethal	Palmer & Radeleff(1964)
PGBE ester	Cow	50 mg/kg	8	No effect	Palmer, et. al (1964)
PGBE ester	Cow	25 mg/kg	20	No effect	Palmer, et. al (1964)
PGBE ester	Sheep	25 mg/kg	10	No effect	Palmer & Radeleff(1969)
PGBE ester	Chicken	100 mg/kg	10	Small weight loss	Palmer & Radeleff(1969)
PGBE ester	Chicken	250 mg/kg	10	Greater weight loss	Palmer & Radeleff(1969)

PHENOXY HERBICIDES AS TERATOGENS, MUTAGENS CARCINOGENS AND COMMENTS ON DIOXIN

Specific tests to determine the biological potential of chemicals as teratogens, mutagens on carcinogens are outlined by Mrak (1969). The techniques employed frequently involve high doses, extended periods of e.posure, force feeding, subcutaneious injection, exotic solvents and inbred strains of laboratory animals. Such techniques bear little resemblance to the exposure non-target organisms encounter due to field use of chemicals. These tests only establish that chemicals may or may not have the biological potential to induce these effects. Careful interpretation of data is necessary to determine the probability that such effects are likely to occur in the field.

Carcinogenicity

Innes, et al (1969) reports 2,4-D isopropyl ester and 2,4,5-TP yielded an increased tumor incidence in comparison to negative controls but the level of significance was less than 0.02. Mrak (1969) suggests these compounds need more testing but the priority for testing is not high in comparison with some other pesticide.

2,4-D acid, butyl ester, isooctyl ester and 2,4,5-T acid were not tumorgenic in mice (Innis et al. 1969). Mrak (1969) did not find sufficient information on other phenoxy herbicide formulations to make a judgement.

Mutagenicity

2,4-D and 2,4,5-T have mutagenic potential as demonstrated in tests with several plant systems (Mrak, 1969). Unrau and Larter (1952), Unrau (1953, 1954) found "highly significant" abnormalities of chromosome behavior in rapidly dividing cells of wheat and barley sprayed with 2,4-D ethyl ester. Muhling et al (1960) also found chromosomal effects in peas treated with 2,4-D. Anderson (1967) on the other hand used a histidine deficient mutant of Salmonella to look for mutagenic effects of many chemicals. While several known mutagens induced mutations in his test, none of 120 herbicides tested did so. Similar results were found in tests with other organisms using a similar strategy (Anderson, 1967). The likelihood of significant mutagenesis occurring from normal use of phenoxy herbicides is small.

Terratogenicity

Mrak (1969) summarized the Bionetics research data on terratogenicity of herbicides.

2,4-D isoctyl ester, 2,4-D butyl ester and 2,4-D isopropyl ester produced statistically significantly higher incidences of congenital malformations in mice or rats. 2,4-5-T was intensively examined in the Bionetics study because it proved highly teratogenic. Mrak (1969) also details test results from Bionetics which show many of these same formulations are not teratogenic in other strains of mice or rats or when other means of exposure are used. Macleod et. al. (1971) questions the adequacy of the Bionetics data because known teratogens and embryo toxins failed to

produce significant effects in these tests. The contamination of the Bionetics 2,4,5-T with high levels of 2,3,7,8- tetrachlorodibenzo-p-Dioxin (dioxin) further invalidates the data for 2,4,5-T.

Verrett (1970) reported 2,4-D, 2,4,5-T and 2,4,5-TP all produced terrata and chick edema syndrome following injection into the yolk sac of fertile chicken eggs. This is an extremely sensitive test and the degree to which it can be extrapolated to field exposure is limited. Johnson (1971) summarized a variety of tests for teratogenicity of the phenoxy herbicides. The studies with presently available commercial formulations of 2,4,5-T and 2,4-D show no teratogenic effects in rats at rates up to 50 mg/kg/day and 87.5 mg/kg/day respectively. Some fetal resorptions appear at higher levels. Tests with silvex (up to 100 mg/kg/day) showed no effects in rats. Higher ratio caused fetel resorptions or maternal toxicity.

Sparschu, Dunn, and Rowe (1971) determined the teratogenic properties of dioxin. Their findings suggest the earlier findings of 2,4,5-T teratogenicity may be attributed to dioxin contamination of 2,4,5-T.

The National Academy of Sciences Advisory Committee on 2,4,5-T wrote the following in their report to the Environmental Protection Agency.

> "Much of the general toxicity attributed to 2,4,5-T in the past now appears to have been caused by the contaminant TCDD (dioxin). The herbicide when essentially free of this contaminant, e.g. 1 ppm, has relatively low toxicity for all animal forms in which it has been tested.

"Particular attention was given to the teratogenic potential of both 2,4,5-T and TCDD. Acceptable data are now available on the embryotoxicity of 2,4,5-T in 6 mammalian species, mouse, rat, hamster, rabbit, sheep and rhesus monkey. None of these showed adverse effects at dosage of 40 mg/kg/day of maternal weight.

The mouse appears to be more sensitive than the other forms studied in that it shows a low level of teratogenicity (cleft palate) at 100 mg/kg/day given throughout organogenesis, whereas hamster and rat required higher dosage to obtain comparable effects. It is likely that all species could be caused to show some embryotoxicity if 2,4,5-T dosage were raised high enough, a fact already known for many prevalent environmental chemicals such as aspirin, caffein, nicotine and organic mercury.

The dioxin contaminant TCDD also has been shown to have a low teratogenic potential at doses in excess of 0.001 mg/kg, but this dosage level is virtually impossible with currently produced 2,4,5-T. No evidence has been found of significant potentiative interaction between 2,4,5-T and TCDD."

The dioxin content of phenoxy herbicides is important. Dichlorodibenzo-p-dioxin would be the major species of dioxin in 2,4-D. The tetrachloro-dibenzo-p-dioxin would be the major species of dioxin in both 2,4,5-T and 2,4,5-TP. Johnson (1971) reports eight lots of silvex from production run

material (1967, 1968, and 1969 lots) did not contain detectable quantities of dioxine. Current 2,4,5-T contains less than 0.5 ppm dioxin. Dichlorodibenzo-p-dioxin is not formed in the manufacture of 2,4-D. Kearney et al (1970) analyzed 129 samples of 18 chlorophenol based pesticides for dioxin. Only occasional samples of 2,4,5-T contained more than 0.5 ppm dioxin. No samples collected after June 1970 contained more than 0.5 ppm dioxin.

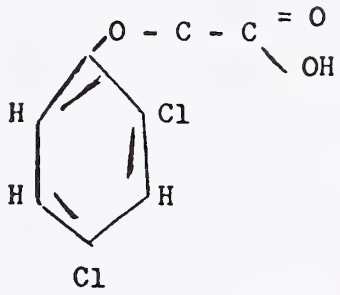
Kearney et al (1970) reported the behavior of dioxin in the environment. They found no uptake of dioxin from soil by plants and no translocation of dioxin from treated foliage. Dioxin residues may be subject to weathering. Dioxin is persistent in soil but does not leach in the soil profile. It is probably tightly bound by soil components. Dioxin is subject to photodecomposition but the significance of this in the field is questionable.

Johnson (1970) reports dioxin is not likely to concentrate in fats like DDT. When 2,4,5-T treated paper or foliage is burned, no dioxin was detected in the vapor phase.

Physical Properties

References

2,4-D Acid

specific gravity	1.57	Bailey and White (1965)
melting point	139°C	" "
solubility in H_2O	725 ppm @25°C	" "
structure:		" "

boiling point 160°C @0.4mm Hg — Melnikov 1971

melting points and solubilities of several salts of 2,4-D and melting and boiling points of several esters of 2,4-D are given by Melnikov (1971).

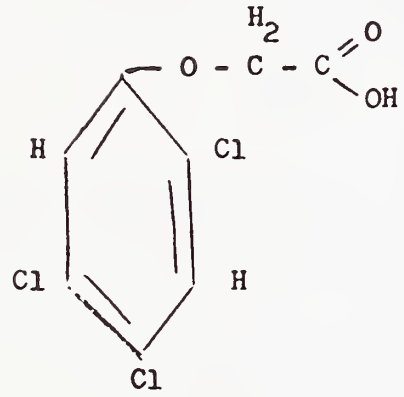
vapor pressure of 2,4-D esters is difficult to measure and there is little agreement on values.

ester	Vapor pressure(mm Hg @25°C)	References
Isopropyl	4.6×10^{-5}	Flint et al (1968)
Isopropyl	10.5×10^{-3}	Warren & Gillis(1952)
Butyl	8.9×10^{-6}	Hamaker & Kerlinger (1969)
Isooctyl	2×10^{-6}	

ester	Vapor pressure (mm hg @25°C)	References
Ethlhexyl	2×10^{-6}	Flint et al (1968)
PGBE	3×10^{-6}	" " "
Butoxyethanol	4.5×10^{-6}	" " "

2,4,5-T

Structure:



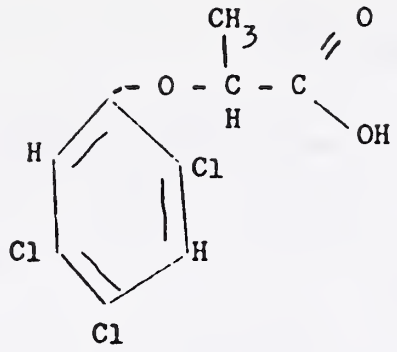
Bailey & White(1965)

specific gravity	1.80	" " "
melting point	154	" " "
solubility in H_2O	280 ppm @25° C	" " "

The melting points and water solubilities of several salts and melting and boiling points of some esters of 2,4,5-T are given by Melnikov (1971). Vapor pressures of 2,4,5-T esters will be similiar to vapor pressure of corresponding ester of 2,4-D. Generally speaking, ethyl, propyl, isopropyl, butyl and isobutyl esters are high volatile, whereas heavier esters such as PGBE, ethylhexyl, isooctyl, or butoxyethanol are low volatile.

2,4,5-TP

Structure:



References

Bailey & White (1965)

Melting point	180°C
Solubility in H_2O	140 ppm @25°C
Vapor pressure:	See comments on 2,4,5-T. Same remarks apply to 2,4,5-TP.

LITERATURE CITED

1. Anderson, K. J. 1967. (personal communication) See House, et. al. 1967.
2. Bailey, J. B. and John E. Swift. 1968. Pesticide information and safety manual. Univ. of Calif. Ag. Ext. Service. Berkeley, Calif.
3. Bailey, G. W. and J. L. White. 1965. HERBICIDES: A compilation of their physical, chemical, and biological properties. Residue Reviews 10:97-124.
4. Bjorklund, Nils-Erik and Kurt Erne. 1966. Toxicological studies of phenoxyacetic herbicides in animals. Acta. Vet. Scand. 7:364-390.
5. Butler, P. A. 1965. Effects of herbicides on estuarine fauna. Southern Weed Cont. Conf. Proc. 18:567.
6. Drill, V. A. and T. Hiratzka. 1953. Toxicity of 2,4-D and 2,4,5-T acid. A report on their acute and chronic toxicity in dogs. AMA Arch. Indust. Hyg. Occup. Med. 7:61-7.
7. Edson, E. F., D. N. Sanderson and D. N. Nookes. 1964. Acute Toxicity Data for Pesticides. World Review of Pest Control 4(1) Spring 1965.
8. Flint, G. W., J. J. Alexander and O. P. Funderburk. 1968. Vapor pressures of low volatile esters of 2,4-D. Weed Sci. 16:541-4.
9. Hamaker, J. W. and H. O. Kerlinger. 1969. Vapor pressure of pesticides. In: Pesticide formulation research. Adv. in Chem. Series No. 86. Pages 39-54.
10. Hayes, Wayland J. J. 1963. Clinical handbook on economic poisons. U. S. Dept. Health, Education and Welfare.

11. House, W. B. et. al. 1967. Assessment of ecological effects of extensive or repeated use of herbicides. Final report on Midwest Research Institute Project 3103-B under Dept. of Army Contract DAHC15-68-C-0119.

12. Hughes, J. S. and J. T. Davis. 1963. Variations in toxicity to bluegill sunfish of phenoxy herbicide weeds 11:50-3.

13. Hughes, J. S. and J. T. Davis. 1966. Toxicity of pesticides to bluegill sunfish tested during 1961-1966. Report to Louisiana Wildlife and Fisheries Commission, Monroe, Louisiana.

14. Hunt, L. M., B. N. Gilbert and J. S. Palmer. 1970. Effects of a herbicide, 2-ethylhexyl ester of 2,4-D on magnesium: calcium ratios and blood urea nitrogen levels in sheep and cattle. Bull. Environ. Contamination and Toxicol. 5:54-60.

15. Innes, J. R. M. et. al. 1969. Bioassay of pesticides and industrial chemicals for tumorigenicity in mice: A preliminary note. J. National Cancer Inst. 42:1101-14.

16. Johnson, J. E. 1971. The public health implications of widespread use of the phenoxy herbicides and picloram. Biosci. 21:899-905.

17. Kearney, P. C. et. al. 1970. Report of research on dioxin. ARS, USDA, and PRD to EPA, Beltsville, Maryland.

18. Lawrence, J. N. 1964. Aquatic herbicide data. USDA. ARS, Agricultural Handbook 231.

19. Macleod, C. M. et. al. 1971. Report on 2,4,5-T. A report of the Panel on herbicides of the President's Science Advisory Committee. Office of Science and Technology, Executive Office of the President March 1971.

20. Melnikov, N. N. 1971. Chemistry of pesticides. Residue Reviews. 36:1-480.

21. Mrak, Emil. 1969. Report of the Secretary's Commission on pesticides and their relationship to environmental health. U.S. Dept. HEW. December 1969.

22. Muhling, G. N., J. Van'T Hof, G. B. Wilson and B. H. Grisby. Cytological effects of herbicidal substituted phenols. Weeds. 8:173-181.

23. Mullison, W. R. 1966. Some toxicological aspects of silvex. Southern Weed Cont. Conf. Proc. 19:420-35.

24. Palmer, J. S., D. E. Clark and L. M. Hurt. 1964. Toxicologic effects of silvex on yearling cattle. Am. Vet. Med. Assoc. J. 144:750-755.

25. Palmer, J. S. and R. D. Radeleif. 1964. The toxicologic effects of certain fungicides and herbicides on sheep and cattle. Ann. N.Y. Acad. Sci. 111:729-36.

26. Palmer, J. S. and R. D. Radeleff. 1969. The toxicity of some organic herbicides to cattle, sheep and chickens. Production Research Report No. 106. ARS, USDA.

27. Rowe, V. K. and T. A. Hymas. 1954. Summary of toxicological information on 2,4-D and 2,4,5-T type herbicides and an evaluation of the hazards to livestock associated with their use. Am. J. Vet. Res. 15:622-29.

28. Tucker, R. K. and D. G. Crabtree. 1970. Handbook of toxicity of pesticides to wildlife. Resource Publication No. 84. Bureau of Sport Fisheries and Wildlife. U.S. Dept. of the Interior.

29. Unrau, J. 1953. Cytogenic effects of 2,4-D on cereals. Canadian Seed Growers Assoc. Ann. Report. pages 37-39.

30. Unrau, J. 1954. Cytogenic effects of 2,4-D on cereals. Canadian Seed Growers Assoc. Ann. Report. pages 25-28.

31. Unrau, J. and E. N. Larter. 1952. Cytogenical responses of cereals to 2,4-D Canadian J. Bot. 30:22-27.

32. Verrett, J. 1970. Testimony before the U.S. Senate Committee on Commerce, Sub-Committee on Energy, Water, Natural Resources and the Environment. 15 April 1970. Serial 91-60. Pages 190-203.

33. Warren, J. C. R. and A. Gillies. 1952. Proc. 6th Meeting Eastern Soc., National Weed Committee. page 98.

Section III METABOLISM OF 2,4-D, 2,4,5-T, and 2,4,5-TP 1/

2,4-D [2,4-Dichlorophenoxyacetic acid]

In feeding studies of 2,4-D with dairy cows and steers, (12, 13, 48, 49, 65) 2,4-D was found unchanged in the urine only. No evidence of betaoxidation was found. Similar findings were obtained with sheep. Ninety-six percent of an orally administered dose of 2,4-D-C-^{14} to a sheet was excreted unchanged in the urine in 72 hours and slightly less than 1.4% in the feces. Very little residual radioactivity was found in edible tissue (28).

In rats receiving 1 to 10 mg of 2,4-D, there was almost complete excretion of the herbicide in the urine and feces in 48 hours. At higher dosage levels, some accumulation in tissues occurred. Analyses also indicated that traces of an unidentified metabolite appeared in the urine (60).

After exposure of bean plants (Phaseolus), sun flowers (Helianthus annus), maize (Zea mays) or barley (Hordeum) to 2,4-D, 2,4-dichlorophenol was observed (111).

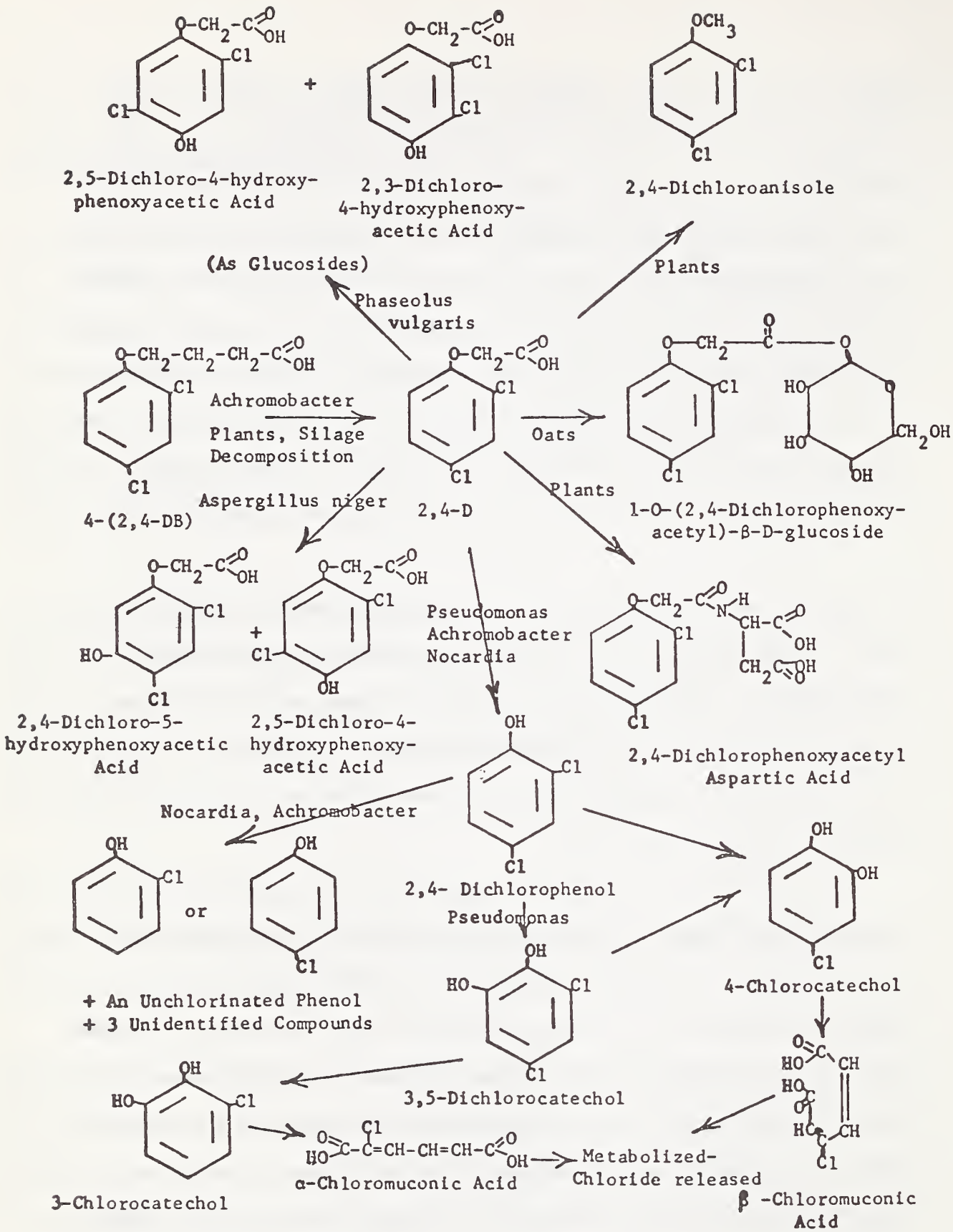
Hydrolysis of esters (30, 75) and decarboxylation (14, 33, 81) of 2,4-D by plants has also been shown. The free acid has been demonstrated on bean plants, corn plants and forage after treatment with 2,4-D butoxyethanol, propylene glycol butyl, butyl and 2-ethylhexyl

esters (39, 51, 53, 63). Treatment of lemons with C^{14} labeled 2,4-D isopropylester indicated that the ester was hydrolyzed and that part of the 2,4-D then reacted with some plant constituent to form an ester-like complex. Ester-like residues were also found after treatment with the sodium, diethanolamine, or triethanolamine salts (34, 35). Samples of fresh citrus peel were prepared by compositing peel samples obtained from oranges from trees sprayed with 2,4-D isopropyl ester. In addition to free acid and ester, a conjugate was also found. The latter became available for extraction only after heat treatment. Preliminary investigations indicated that 2,4-D was conjugated with pectin (73).

On cotton, cucumbers, beans, and grain sorghum, labeled 2,4-D gave rise to $C^{14}O_2$ (56, 106, 109). Pea and tomato plants have also been studied (38). In young leaves and bolls of cotton, material chromatographically different from 2,4-D was formed. Sorghum converted 2,4-D to a complex different than that found in cotton (74, 77, 90, 104, 105, 106, 107, 108, 109).

Amino acids have been implicated in the formation of some compounds, as in the case of 2,4-dichlorophenoxyacetylaspartic acid (3, 11). Evidence indicated that 2,4-D moved through plants as a protein complex, which could be recovered after aqueous extraction and NaOH hydrolysis, into the roots where most of the degradation occurred (22). Resistant plants were grown in water cultures treated with 2,4-D. Leaves were homogenized and a protein fraction was obtained that contained 2,4-D

1/ The numbers in parenthesis refer to references at the end of the metabolism section.


2,5-Dichloro-4-hydroxyphenoxyacetic Acid
2,3-Dichloro-4-hydroxyphenoxyacetic Acid
2,4-Dichloroanisole
(As Glucosides)
Phaseolus vulgaris
Plants
4-(2,4-DB)
Achromobacter
Plants, Silage Decomposition
Oats
Aspergillus niger
2,4-D
Plants
1-O-(2,4-Dichlorophenoxyacetyl)-β-D-glucoside
2,4-Dichloro-5-hydroxyphenoxyacetic Acid
2,5-Dichloro-4-hydroxyphenoxyacetic Acid
Pseudomonas
Achromobacter
Nocardia
2,4-Dichlorophenoxyacetyl Aspartic Acid
Nocardia, Achromobacter
2,4- Dichlorophenol
Pseudomonas
or
+ An Unchlorinated Phenol
+ 3 Unidentified Compounds
4-Chlorocatechol
3,5-Dichlorocatechol
3-Chlorocatechol
α-Chloromuconic Acid
Metabolized-Chloride released
β -Chloromuconic Acid

in a bound form not further identified (24). In big leaf maple (Acer Macrophyllum Pursh), 2,4-D was converted into two metabolites. One of these was the same compound characterized previously (18) as a 2,4-D protein complex which yielded 2,4-D and 12 amino acids on acid hydrolysis (80).

Glucose esters were suggested (31, 61, 62) and studies have shown that glucosidic complexes were formed. From stem tissues of oats (Avena sativa), 1-O-(2,4-dichlorophenoxyacetyl)-B-D-glucose was isolated (97), and from stems of the kidney bean (Phaseolus vulgaris), the 2,5- and 2,3-dichlorophenoxyacetic acid glucosides have been obtained (96).

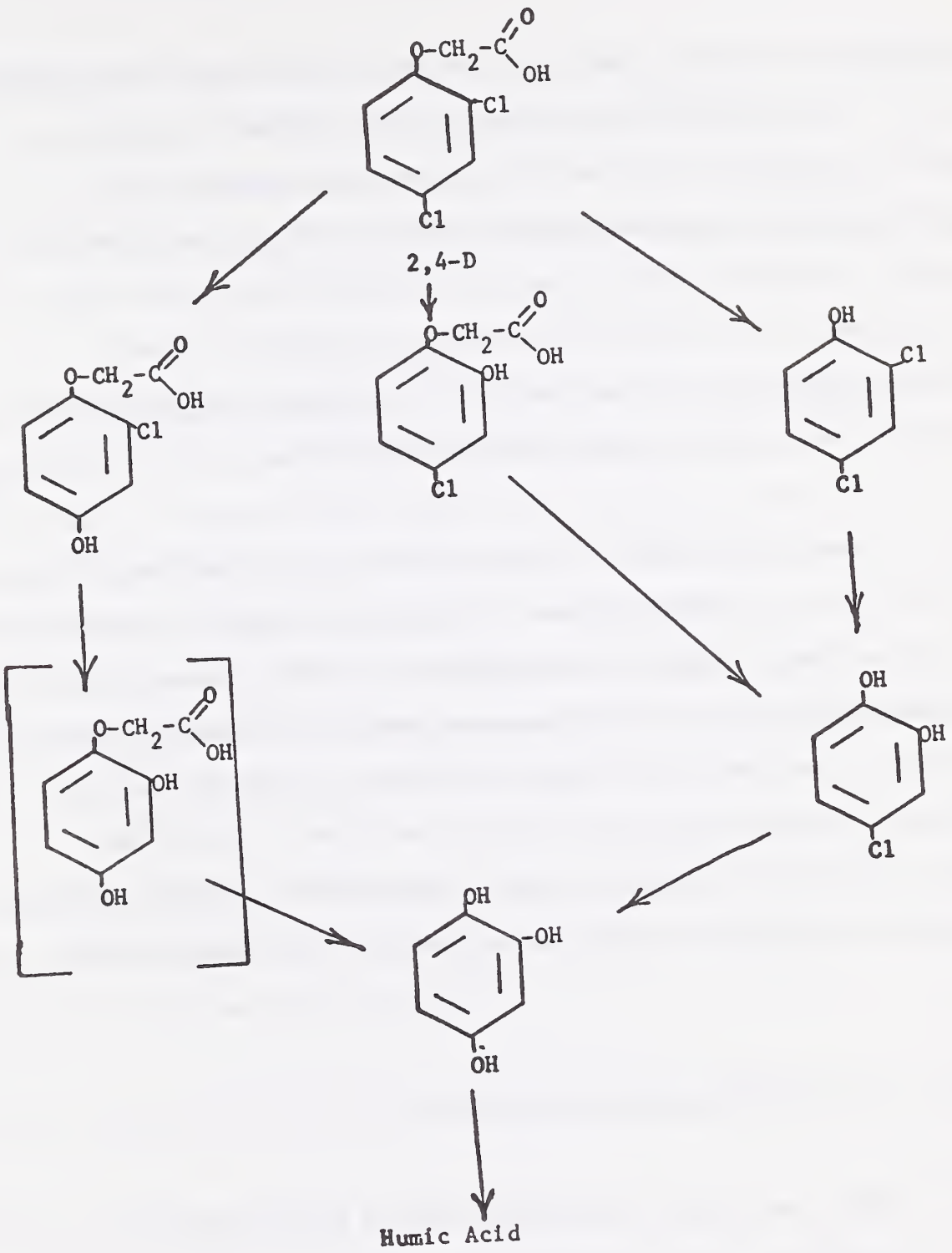
From comparative studies with sensitive and insensitive plants, two metabolic paths were proposed involving initial glucose ester formation and oxidation ring cleavage of the aromatic ring in yield monochloroacetic acid (100). The latter has been detected in plants prior to the onset of treatment symptoms; and it has been suggested that the effect of 2,4-D resulted from the action of monochloroacetate arising from 2,4-D degradation (100, 112).

Plants are capable of hydroxylating phenoxyacetic acids (95, 110). When bean plants were treated with 2,4-D, three compounds were found (31). One corresponded roughly to that of 2,4-dichloranisole; one was water-soluble, ether-insoluble ester derivative; and the third, an ether-soluble compound with a basic structural change. The methyl derivative was less volatile than 2,4-D methyl ester, but more volatile than the 4-hydroxy-2,4-D methyl

ester. It might be one of the other two hydroxy derivatives; however, 6-hydroxy-2,4-D was not detected (10, 37, 55, 57, 58).

The biotransformation of 2,4-dichlorophenoxyalkanoic acids and related compounds by soil microflora has been extensively studied (4, 5, 6, 7, 9, 15, 19, 20, 21, 29, 36, 37, 40, 43, 54, 59, 78, 91, 101, 102, 103). Phenoxyalkanoic acids with an even number of carbons in the fatty acid were converted by B-oxidation to products with an even number of carbons (47, 50, 64, 101, 102, 103). A second mechanism involved cleavage of the ether linkage (8, 17, 22, 70, 71, 72).

Evidence has been obtained that 2,4-D is dissimilated by a variety of microorganisms (1, 82) through a 2,4-dichlorophenol and 4-chlorocatechol (7). A product from the degradation of 2,4-D by bacteria of the genus Pseudomonas has been identified as B-chloromuconic acid. A second species of Pseudomonas gave rise to a-chloromuconic acid (44). In other studies, 6-hydroxy-2,4-D was reported (67). Pure cultures of a Nocardia species and an Achromobacter strain of bacteria rapidly degraded 2,4-D and the presence of 2,4-dichlorophenol, chlorohydroquinone, a monochlorophenol, an unchlorinated phenol and three other unidentified compounds have been demonstrated (6, 15, 16, 40, 78, 92, 93, 94). The main product of 2,4-D metabolism by the mold Asperigillus niger van Tiegh was 2,4-dichloro-5-hydroxyphenoxyacetic acid. By means of infrared and mixed melting points, a second metabolite was identified as the 2,4-dichloro-4-hydroxyphenoxy-acetic acid--the first time such a rearrangement was reported, Another unidentified acid, not the 3- or 6- hydroxyacid, was also found (41, 42).


2,4-D
Humic Acid

Arthrobacter sp. degraded 2,4-D via 2,4-dichlorophenol and 2,3-dichloroanisole (68, 69). In excess of 80% of the chloride was released in a 3 hour incubation period with crude extracts or the soluble fraction (67). A Corynebacterium species also degraded 2,4-D with quantitative release of chloride. In natural surface waters, 2,4-D isopropyl and butyl esters were hydrolyzed to 2,4-D and their respective alcohols (2). When triethanolamine salts of C^{14}-carboxy labeled 2,4-D were applied in water to forest litter, liberation of $C^{14}O2$ was rapid (79).

In the presence of water and ultraviolet light, 2,4-D decomposed rapidly with formation of 2,4-dichlorophenol. This underwent further decomposition to 4-chlorocatechol, polymeric humic acids and chloride. Some 2-hydroxy-4-chlorophenoxyacetic acid and a very small amount of 2-chloro-4-hydroxy phenoxyacetic acid were present (32, 52, 98, 99). In the presence of riboflavin, compounds containing more than one aromatic nucleus were probably also formed in addition to 2,4-dichlorophenol. Products differed according to the original pH and concentration of the treated solution (52).

2,4,5-T [2,4,5-Trichlorophenoxyacetic Acid]

Cows fed 2,4,5-T excreted it as a soluble salt in their urine (83).

When Winesap and Staymen Winesap cultivars were exposed to 2,4,5-T some decarboxylation occurred (81). Bean plants (Phaseolus), sun flowers (Helianthus annus) and barley (Hordeum) converted 2,4,5-T to its phenol (111). 2,4,5-T was also decarboxylated by woody plants (14).

From comparative studies with sensitive and insensitive plants, two paths were proposed, involving initial glucose ester formation and oxidative ring cleavage to yield monochloroacetate. The latter was detected in treated plants prior to the onset of treatment symptoms (100).

Sweetgum (Liquidambar styraciflua L.) and southern red oak (Quercus falcata Michx.) were sprayed with an aqueous homogenate of 2,4,5-T n-butyl ester. After one month, leaves were collected and assayed using gas chromatography to detect residues. 2,4,5-Trichlorophenol was observed but no evidence was found to indicate formation of 2,4,5-Trichloranisol (45, 46). After application to Bigleaf maple (Acer Macrophyllum Pursh) and mesquite seedlings, 2,4,5-T was metabolized but the products were not identified (76, 80).

Triethanolamine salts of C^{14}-carboxy labeled 2,4,5-T were applied in water to the surface of some collected forest litter. Liberation of $C^{14}O_2$ was slow but increased with time (79).

2,4,5-TP (Silvex) [2-(2,4,5-Trichlorophenoxy)propionic Acid]

When fed to cows, silvex was excreted as a soluble salt in the urine. Kuron, the propylene glycol butyl ether ester of silvex, was hydrolyzed prior to elimination (83).

Samples of fresh citrus peel were prepared by compositing peel samples obtained from oranges from trees sprayed with 2,4,5-TP propylene glycol butyl ether ester. In addition to free acid and ester, a conjugate was found. The latter became available for extraction only after heat treatment. Preliminary investigations indicated that 2,4,5-TP was conjugated with pectin (73). Decarboxylation of 2,4,5-TP by prickly pear (Opuntia spp.) was 1/2 to 1/3 of that by soybean. In addition to unaltered 2,4,5-TP, at least four labeled metabolites were observed after application of silvex-1-C^{14} was applied to prickly pear (26, 27).

When the propylene glycol ether ester was applied to water overlying various soil types, the herbicide was hydrolyzed almost totally to the acid in about two weeks. Absorption of the acid by the soils was also indicated (116).

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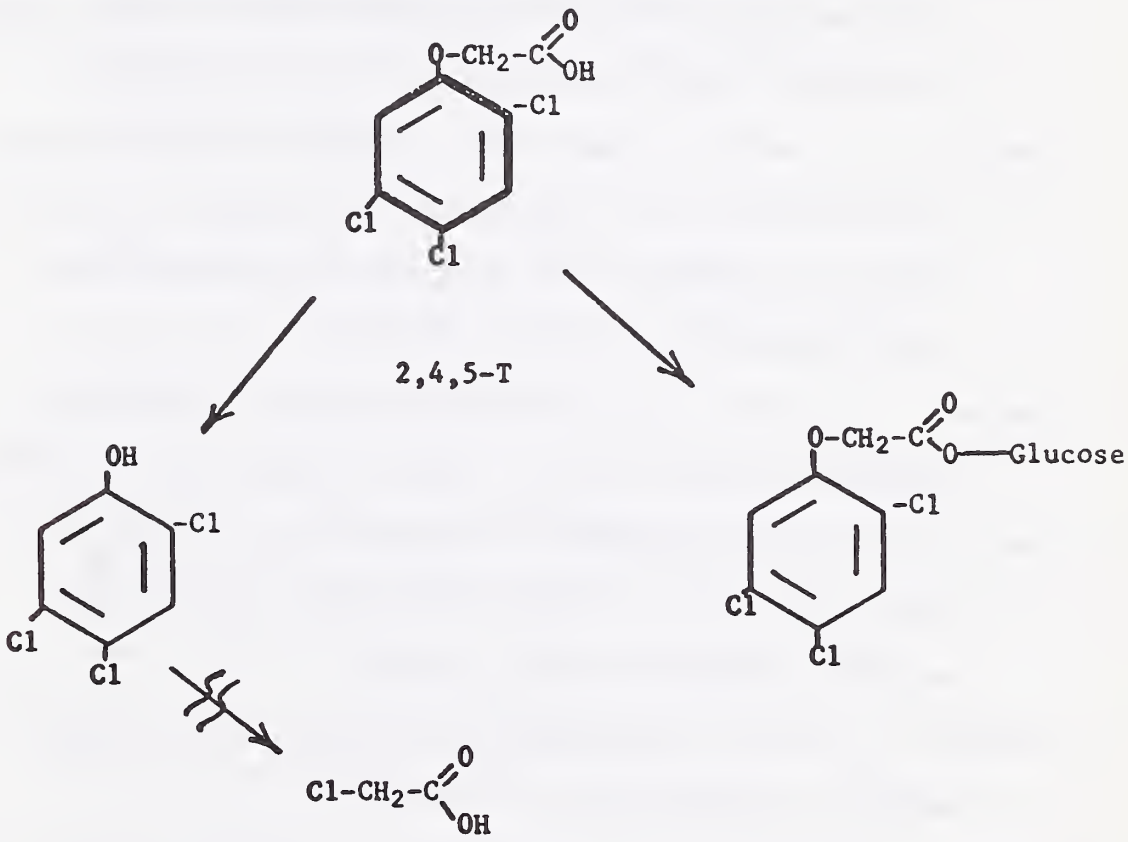

monochloroacetate

REFERENCES TO METABOLISM OF
2,4-D, 2,4,5-T, 2,4,5-TP

1. Alexander, M. and M. I. H. Aleem, 1961. Effect of Chemical structure on microbial decomposition of aromatic herbicides. Journal of Agriculture and Food Chemistry, vol. 9, p. 44-47.

2. Aly, Osman M. and Samuel D. Faust, 1964. Studies on the fate of 2,4-D and ester derivatives in natural surface water. Journal of Agriculture and Food Chemistry, vol. 12, p. 541-546.

3. Andreae, W. A. and N. E. Good, 1957. Studies on 3-indole acetic acid metabolism. IV. Conjugation with aspartic acid and ammonia as processes in the metabolism of carboxylic acid Plant Physiology, vol. 32, p. 566-572.

4. Audus, L. J., 1949. Biological detoxification of 2,4-dichlorophenoxyacetic acid in soil. Plant and soil, vol. 2, p. 31-36.

5. Audus, L. J., 1950. Biological detoxification of 2,4-dichlorophenoxyacetic acid in soils: isolation of an effective organism. Nature, vol. 166, p. 356.

6. Audus, L. J., 1951. The biological detoxification of hormone herbicides in soil, vol. 3, p. 170-192.

7. Audus, L. J., 1952. The decomposition of 2,4-dichlorophenoxyacetic acid and 2-methyl-4-chlorophenoxyacetic acid in the soil. Journal of the Science of Food and Agriculture, vol. 3, p. 268-274.

8. Audus, L. J., (Editor), 1964. The herbicide behavior. In the Physiology and Biochemistry of Herbicides, Academic Press, p. 75-206.

9. Audus, L. J. and K. V. Symonds, 1955. Further studies on the breakdown of 2,4-dichlorophenoxyacetic acid by a soil bacterium. Annals of Applied Biology, vol. 42, p. 174-182.

10. Bach, M. K., 1961. Metabolites of 2,4-dichlorophenoxyacetic acid from bean stems. Plant Physiology, vol. 36, p. 558-565.

11. Bach, M. K. and J. Fellig, 1961. Metabolism of carboxyl-C^{14}-labeled 2,4·dichlorophenoxyacetic acid by bean stems: heterogeneity of ehtanolsoluble, ether-soluble products. Nature, volume 189, p. 763.

12. Bache, C. A., D. D. Hardee, R. F. Holland, and D. J. Lisk, 1964. Absence of phenoxyacid herbicide residues in the milk of dairy cows at high feeding levels. Journal of Dairy Science, vol. 47, p. 298-299.

13. Bache, C. A., D. J. Lisk, D. G. Wagner, and R. G. Wagner, 1964. Elimination of 2-methyl-4-chlorophenoxyacetic acid and 4-(2-methyl-4-chlorophenoxybutyric) acid in the urine from cows. Journal of Dairy Science, vol. 47, p. 93-95.

14. Basler, E., 1964. The decarboxylation of phenoxyacetic acid herbicides by excised leaves of woody plants. Weeds, vol. 12, p. 14-16.

15. Bell, G. R., 1957. Some morphological and biochemical characteristics of a soil bacterium which decomposes 2,4-dichlorophenoxyacetic acid. Canadian Journal of Microbiology vol. 3, p. 821-840.

16. Bell, G. R., 1960. Studies on a soil achromobacter which degrades 2,4-dichlorophenoxyacetic acid. Canadian Journal of Microbiology, vol. 6, p. 325-337.

17. Bocks, S. M., J. R. L. Smith, and R. O. C. Normal, 1964. Hydroxylation of phenoxyacetic acid and anisole by Aspergillus niger. (van Tiegh). Nature, vol. 201, p. 398.

18. Butts, J. S., and S. C. Fang, 1956. Tracer studies on the mechanisms of action of hormonal herbicides. U.S. Atomic Energy Report TID 7512, p. 209-214.

19. Byrde, R. J. W., F. Harris, and D. Woodcock, 1956. The metabolism of w-(2-napthyloxy)-n-alkyl-carboxylic acids by Aspergillus niger. Biochemical Journal, vol. 64, p. 154-160.

20. Byrde, R. J. W., D. Woodcock, 1957. 2. The metabolism of some phenoxy-n-alkyl-carboxylic acids by Aspergillus niger. Biochemical Journal, vol. 65, p. 682-686.

21. Byrde, R. J. W., and D. Woodcock, 1958. 3. The metabolism of w-(2-napthyloxy)-n-alkyl-acrboxylic acids by Sclerotinia laxa. Biochemical Journal, vol. 69, p. 19-21.

22. Canny, M. J., and K. Markus, 1960. The breakdown of 2,4-dichlorophenoxyacetic acid in shoots and roots. Australian Journal of Biological Sciences, vol. 13, p. 486-500.

23. Castlefranco, Paul A. Oppenheim, and Shogo Yamaguchi, 1963. Riboflavin mediated photodecomposition of amitrole in relation to chlorosis. Weeds, vol. 11, p. 111-115.

24. Chkanikov, D. I. and N. N. Pavlova, 1966. Proteins responsible for 2,4-D detoxication in resistant plants. Agrokhimiya, No. 5, p. 115-119.

25. Chow, P. N., 1966. Absorption, Translocation and Metabolism of 2-(2,4,5-Trichlorophenoxy) propionic Acid-1-C^{14} in Opuntia spp. Dissertation Abstracts, vol. 26, p. 4152.

26. Chow, P. N., O. C. Burnside, T. L. Lavy, and H. W. Knoche, 1966. Absorption, Translocation and Metabolism of Silvex in Prickly Pear. Weeds, vol. 14, p. 38-41.

27. Clark, D. E., J. E. Young, R. L. Younger, L. M. Hunt, and J. K. McLaren, 1964. The fate of 2,4-dichlorophenoxyacetic acid in sheep. Journal of Agriculture and Food Chemistry vol. 12, p. 43-45.

28. Clifford, D. R., and D. Woodcock, 1964. Metabolism of phenoxyacetic acid by Aspergillus niger van Tiegh. Nature, vol. 203 p. 763.

29. Crafts, A. S., 1960. Evidence for hydrolysis of 2,4-D during absorption by plants. Weeds, vol. 8, p. 19-25.

30. Crosby, D. G., 1964. Metabolites of 2,4-dichlorophenoxyacetic acid (2,4-D) in bean plants. Journal of Agriculture and Food Chemistry, vol. 12, p. 3-6.

31. Crosby, D. G., and Helmut O. Tutass, 1966. Photodecomposition of 2,4-Dichlorophenoxyacetic acid. Journal of Agriculture and Food Chemistry, vol. 14, p. 596-599.

32. Edgerton, L. J. and M. B. Hoffman, 1961. Fluorine substitution affects decarboxylation of 2,4-dichlorophenoxyacetic acid in apple. Science, vol. 134, p. 341.

33. Erickson, L. C., B. L. Brannaman, and Charles W. Coggins, Jr., 1963. Residues in stored lemons treated with various formulations of 2,4-D. Journal of Agriculture and Food Chemistry, vol. 11, p. 437-440.

34. Erickson, L. C., and Henry Z. Nield, 1962. Determination of 2,4-dichlorophenoxyacetic acid in citrus fruit. Journal of Agriculture and Food Chemistry, vol. 10, p. 204-207.

35. Evans, W. C., and P. Moss, 1957. The metabolism of the herbicide p-chlorophenoxyacetic acid by a soil microorganism - the formation of a B-chloro-muconic acid on ring fission. Biochemical Journal, vol. 65, p. 8P.

36. Evans, W. C., and B. S. W. Smith, 1954. The photochemical inactivation and microbial metabolism of the chlorophenoxy-acetic acid herbicides. Biochemical Journal, vol. 57, p. xxx.

37. Fang, S. C., 1958. Absorption, translocation, and metabolism of 2,4-D-1-C^{14} in pea and tomato plants. Weeds, vol. 6, p. 179-186.

38. Fang, S. C., E. G. Jaworski, A. V. Logan, V. H. Freed, and J. S. Butts, 1951. The absorption of radioactive 2,4-dichloro-phenoxyacetic acid and the translocation of C^{14} by bean plants Archives of Biochemistry and Biophysics, vol. 32, p. 249-255.

39. Faulkner, J. K. and D. Woodcock, 1961. Fungal detoxication V. Metabolism of o- and -p-chlorophenoxyacetic acids by Aspergillus niger. Journal of the Chemical Society (London) p. 5397-5400

40. Faulkner, J. K. and D. Woodcock, 1964. Metabolism of 2,4-dichlorophenoxyacetic acid (2,4-D) by Aspergillus niger van Tiegh. Nature, vol. 203, p. 865.

41. Faulkner, J. K. and D. Woodcock, 1965. Fungal detoxication. VII. Metabolism of 2,4-dichloro-phenoxy-acetic and 4-chloro-2-methylphenoxyacetic acids by Aspergillus niger Journal of the Chemical Society, p. 1187-1191.

42. Fawcett, C. H., J. M. A. Ingram, and R. L. Wain, 1954. The B-oxidation of w-phenoxyalkylcarboxylic acids in the flax plant in relation to their plant growth-regulating activity. Proceedings of the Royal Society of London, vol. 142B, p. 60-72.

43. Fernley, H. N. and W. C. Evans, 1959. Metabolism of 2,4-dichlorophenoxyacetic acid by soil Pseudomonas: isolation of a-chloromuconic acid as an intermediate. Biochemical Journal, vol. 73, p. 22P.

44. Fitzgerald, C. H., 1966. The degradation of 2,4,5-Trichlorophenoxy-acetic acid in woody plants. Dissertation abstracts, vol. 27B, p. 1772.

45. Fitzgerald, Charles H., Claud L. Brown, and Edwin G. Beck, 1967. Degradation of 2,4,5-Trichlorophenoxyacetic acid in woody plants. Plant physiology, vol. 42, p. 459-460.

46. Gutenmann, W. H., 1964. Conversion of 4-(2,4-DB) to 2,4-dichloro-phenoxycrotonic acid (2,4-DC) and production of 2,4-D from 2,4-DC in soil. Journal of Agriculture and Food Chemistry, vol. 12, p. 322-323.

47. Gutenmann, W. H. D. D. Hardee, R. F. Holland, and D. J. Lisk, 1963. Disappearance of 4-(2,4-dichlorophenoxybutyrick) acid herbicide in the dairy cow. Journal of Dairy Science vol. 46, p. 991-992.

48. Gutenmann, W. H., D. D. Hardee, R. F. Holland, and D. J. Lisk, 1963. Residue studies with 2,4-dichlorophenoxyacetic acid herbicide in the dairy cow and in a natural and artificial rumen. Journal of Dairy Science, vol. 46, p. 1287, 1288.

49. Gutenmann, W. H., M. A. Loos, M. Alexander, and D. J. Lisk, 1964. Beta oxidation of phenoxyalkanoic acids in soil. Soil Science Society of America Proceedings, vol. 28, p. 205-207.

50. Hagen, C. E., C. O. Clagett, and E. A. Helgeson, 1949. 2,4-dichlorophenoxyacetic acid inhibition of castor bean lipase. Science vol. 110, p. 116-117.

51. Hansen, J. R. and K. P. Buchholtz, 1952. Inactivation of 2,4-D by riboflavin in light. Weeds, vol. 1, p. 237-242.

52. Hay, J. R. and K. V. Thiman, 1956. The fate of 2,4-dichlorophenoxyacetic acid in bean seedlings. I. Recovery of 2,4-dichlorophenoxyacetic acid and its breakdown in the plant. Plant physiology, vol. 31, p. 382-387,

53. Henderson, M. E. K., 1957. Metabolism of methoxylated aromatic compounds by soil fungi. Journal of General Microbiology, vol. 16, p. 686-695.

54. Holley, R. W., 1952. Studies of the fate of radioactive 2,4-dichlorophenoxyacetic acid in bean plants. II. A water soluble transformation product of 2,4-D. Archives of Biochemistry and Biophysics, vol. 35, p. 171-175.

55. Holley, R. W., F. P. Boyle, and D. B. Hand, 1950. Studies on fate of radioactive 2,4-D in bean plants. Archives of Biochemistry and Biophysics, vol. 27, p. 143-151.

56. Jaworski, E. G., and J. S. Butts, 1952. Studies in plant metabolism II. The metabolism of C^{14} labeled 2,4-dichlorophenoxyacetic acid in bean plants. Archives of Biochemistry and Biophysics vol. 38, p. 207-218.

57. Jaworski, E. G., S. C. Fang, and V. H. Freed, 1955. Studies in plant metabolism. V. The metabolism of radioactive 2,4-D in etiolated bean plants. Plant Physiology, vol. 30, p. 272-275.

58. Jensen, H. L., and H. I. Peterson, 1952. Decomposition of hormone herbicides by bacteria. Acta Agriculturae Scandinavica, vol. 2, p. 215-231.

59. Khanna, S., and S. C. Fang, 1966. Metabolism of C^{14}-Labeled 2,4-Dichlorophenoxyacetic Acid in rats. Journal of Agricultural and Food Chemistry, vol. 14, p. 500-503.

60. Klambt, H., 1961. Wachstumsinduktion und Wuchsstoffmetabolismus im Weizenkoleoptilzylinder. Planta. vol. 57, p. 339-353.

61. Klambt. H., 1961. Wachstumsinduktion und Wuchsstoffmetabolismus im Weizenkoleoptilzylinder. IV. Die identifizerung eines stoffwechselproduktes der Naphthyl-1-Essigsaure, Das Moglicherweise Beziehung Zur Wachstumsforderung Besitzt. Planta, vol. 57, p. 391-401.

62. Klein, W., F. Korte, I. Weisgerber, R. Kaul, W. Muller and A. Djirsarai 1968. Uber den Metabolismus von Endrin Heptachlor und Telodrin. Qualitas Plantarum et Materiae Vegetabiles, vol. 15, p. 225-239.

63. Levey, Stanley, and Howard B. Lewis, 1947. The metabolism of phenoxyacetic acid, its homologues and some monochlorophenoxyacetic acids. Journal of Biological Chemistry, vol. 168, p. 213-221.

64. Lisk, D. J., Walter H. Gutenmann, Carl A. Bache, Richard G. Warner, and Donald G. Wagner, 1963. Elimination of 2,4-D in the urine of steers fed 4-(2,4-DB) or 2,4-D. Journal of Dairy Science, vol. 46, p. 1435-1437.

65. Loos, M. A., 1967. Decomposition of 2,4-D and related phenoxyacetic acids by an Arthrobacter species. Dissertation Abstracts, vol. 27B, p. 4232-4233.

66. Loos, M. A., J. M. Bollag, and M. Alexander, 1967. Phenoxyacetate herbicide detoxication by bacterial enzymes. Journal of Agricultural and Food Chemistry, vol. 15, p. 858-860.

67. Loos, M. A., R. N. Roberts, and M. Alexander, 1967. Phenols as Intermediates in the decomposition of phenoxyacetates by an arthrobacter species. Canadian Journal of Microbiology vol. 13, p. 679-690.

68. Loos, M. A., R. N. Roberts, and M. Alexander, 1967. Formation of 2,4-Dichlorophenol and 2,4-Dichloroanisole from 2,4-Dichlorophenoxyacetate by Arthrobacter sp. Canadian Journal of Microbiology, vol. 13, p. 691-699.

69. MacRae, I. C., and M. Alexander, 1963. Metabolism of phenoxyalkyl carboxylic acids by a flavobacterium species. Journal of Bacteriology, vol. 86, p. 1231-1235.

70. MacRae, I. C., and M. Alexander, 1964. Use of gas chromatography for the demonstration of a pathway of phenoxy herbicide degradation. Agronomy Journal, vol. 56, p. 91-92.

71. MacRae, I. C., M. Alexander, and A. D. Rovira, 1963. The decomposition of 4-(2,4-dichlorophenoxy) butyric acid by Flavobacterium sp. Journal of General Microbiology, vol. 32, p. 69-76.

72. Meagher, W. R., 1966. A Heat-Labile Insoluble Conjugated form of 2,4-Dichlorophenoxyacetic acid and 2-(2,4,5-Trichlorophenoxy) propionic acid in citrus peel. Journal of Agricultural and Food Chemistry, vol. 14, p. 599-601

73. Morgan, Page W., and Wayne C. Hall, 1963. Metabolism of 2,4-D by cotton and grain sorghum. Weeds, vol. 11, p. 130-135.

74. Morre, D. James, and B. J. Rogers, 1960. The fate of long chain esters of 2,4-D in plants. Weeds, vol. 8, p. 436-447.

75. Morton, H. L., and R. E. Meyer, 1962. Absorption, translocation and metabolism of 2,4,5-T by mesquite seedlings. Plant Physiology, vol. 37 (Suppl.), p. xxiv-xxv.

76. Nencki, M., and P. Giacosa, 1880. Ueber die oxydation der aromatischen kohlenwasserstoffe in thierkorper. Hoppe-Seylers Zeitschrift fur Physiologische Chemie, vol. 4, p. 325-338.

77. Newman, A. S., and J. R. Thomas, 1950. Decomposition of 2,4-dichlorophenoxyacetic acid in soil and liquid media. Soil Science Society of America Proceedings, vol. 14, p. 160-164.

78. Norris, L. A., 1966. Degradation of 2,4-D and 2,4,5-T in Forest litter. Journal of Forestry, vol. 64, p. 475-476.

79. Norris, L. A. and V. H. Freed, 1966. The metabolism of a series of chlorophenoxyalkyl acid herbicides in bigleaf maple, Acer Macrophyllum Pursh. Weed research, vol. 6, p. 212-220.

80. Primer, P. E., 1967. Investigations into the fate of some C^{14} labeled growth regulators of the phenoxy and naphthalenic types in apple tissue. Dissertation abstracts, vol. 28B, p. 415.

81. Reid, J. J., 1960. Bacterial decomposition of herbicides. Northeastern weed control conference, vol. 14, p. 19-30.

82. St. John, L. E., Jr., D. G. Wagner, and D. J. Lisk, 1964. Fate of atrazine, kuron, silvex, and 2,4,5-T in the dairy cow. Journal of Dairy Science, vol. 47, p. 1267-1270.

90. Slife, F. W., J. L. Key, S. Yamaguchi, and A. S. Crafts, 1962. Penetration, translocation, and metabolism of 2,4-D and 2,4,5-T in wild and cultivated cucumber plants. Weeds, vol. 10, p. 29-35.

91. Steenson, T. I., and N. Walker, 1956. Observations on the bacterial oxidation of chlorophenoxyacetic acids. Plant and soil (Hague), vol. 8, p. 17-32.

92. Steenson, T. I., and N. Walker, 1957. The pathway of breakdown of 2,4-dichloro-and 4-chloro-2-methyl phenoxyacetic acid by bacteria. Journal of General Microbiology, vol. 16, p. 146-154.

93. Steenson, T. I., and N. Walker, 1958. Adaptive patterns in the bacterial oxidation of 2,4-dichloro-and 4-chloro-2-methyl-phenoxyacetic acid. Journal of General Microbiology, vol. 18, p. 692-697.

94. Taylor, H. F., and R. L. Wain, 1962. Side chain degradation of certain ω-phenoxyalkane carboxylic acid by Nocardia coeliaca and other micro-organisms isolated from soil. Proceedings of the Royal Society of London, Series B, vol. 156, p. 172-186.

95. Thomas, E. W., and B. C. Loughman, 1963. Hydroxylation of phenoxy-acetic acid by stem tissue of Avena sativa. Nature, vol. 199, p. 73-74.

96. Thomas, E. W., and B. C. Loughman, 1964. Metabolic fate of 2,4-dichlorophenoxyacetic acid in the stem tissue of Phaseolus vulgaris. Nature, vol. 204, p. 884, 885.

97. Thomas, E. W., B. C. Loughman, and R. G. Powell, 1964. Metabolic fate of some chlorinated phenoxyacetic acids in the stem tissue of Avena Sativa. Nature, vol. 204, p. 286.

98. Tutass, H., 1966. Photodecomposition of 2,4-D. Proceedings 18th Annual California Weed Conference. January 18-20, p. 13-14.

99. Tutass, H. O., and D. G. Crosby, 1965. The photodecomposition of 2,4-D. Abstracts 149th American Chemical Society Meeting, Detroit. April 4-9, p. 11A.

100. Tutass, H. W., and D. G. Crosby, 1968. Relationships among molecular structure, metabolism, and biological activity of halogen-substituted phenoxyacetic acids. Abstracts 155th Meeting American Chemical Society, San Francisco, April 1-5, A-11.

101. Webley, D. M., R. B. Duff, and V. C. Farmer, 1955. Beta-oxidation of fatty acids by Nocardia opaca. Journal of General Microbiology, vol. 13, p. 361-369.

102. Webley, D. M., R. B. Duff, and V. C. Farmer, 1957. Formation of a β-hydroxy acid as an intermediate in the microbiological conversion of monochlorophenoxybutyric acids to the corresponding acetic acids. Nature, vol. 179, p. 1130-1131.

103. Webley, D. M., R. B. Duff, and V. C. Farmer, 1958. The influence of chemical structure on β-oxidation by soil nocardiac. Journal of General Microbiology, vol. 18, p. 735-746.

104. Weintraub, R. L., 1953. Metabolism of 2,4-D by micro-organisms and higher plants. North Central Weed Control Conference Proceedings, vol. 10, p. 6-7.

105. Weintraub, R. L., J. W. Brown, M. Fields, and J. Rohan, 1950. Production of $C^{14}O_2$ from labeled 2,4-dichlorophenoxyacetic acid by plants. American Journal of Botany, vol. 37, p. 682.

106. Weintraub, R. L., J. W. Brown, M. Fields, and J. Rohan, 1952. Metabolism of 2,4-dichlorophenoxyacetic acid. I. $C^{14}O_2$ production by bean plants treated with labeled 2,4-dichlorophenoxyacetic acid. Plant Physiology, vol. 27, p. 293-301.

107. Weintraub, R. L., J. H. Reinhart, and R. A. Scherff, 1956. Role of entry, translocation, and metabolism in specificity of 2,4-D and related compounds. United States Atomic Energy Report of 2,4-D and related compounds. United States Atomic Energy Report TID 7512, p. 203, 208.

108. Weintraub, R. L., J. H. Reinhart, E. A. Scherff, and L. C. Schisler 1954. Metabolism of 2,4-D.III. Metabolism and persistence in dormant plant tissues. Plant Physiology, vol. 29, p. 303-304.

109. Weintraub, R. L., J. N. Yeatman, J. A. Lockhart, J. H. Reinhart, and M. Fields, 1952. Metabolism of 2,4-dichlorophenoxyacetic acid. II. Metabolism of the side chain by bean plants. Archives of Biochemistry and Biophysics, vol. 40, p. 277-285.

110. Wilcox, M., D. E. Moreland, and G. C. Klingman, 1963. Aryl hydroxylation of phenoxyaliphatic acids by excised roots, Physiologia Plantarum, vol. 16, p. 565-571.

111. Chkanikov, D. I., N. N. Pavlova and D. F. Gertsuskii, 1965. Halide-phenols as products of metabolism of halide-phenoxy-acetic acids in plants. Khimiya V Selskom Khozyaistve, vol. 4, pp. 56-60.

112. Crosby, D., 1968. Meeting Report. Pesticides: Metabolism, degradation, and mode of action. Nikko, Japan, August 16-19, 1967. Report submitted by J. E. Casida and K. Fukunaga. Science, vol. 160, pp. 445-450

113. Bollag, J. M., M. A. Loos and M. Alexander, 1967. Enzymatic degradation of phenoxyalkanoate herbicides. Bacteriological proceedings, abstracts, A-42.

114. Bollag, J. M., C. S. Helling and Martin Alexander, 1968. 2,4-D metabolism. Enzymatic hydroxylation of chlorinated phenols. Journal of agriculture and food chemistry, vol. 16, pp. 826-828.

115. Bollag, J. M., C. G. Briggs, J. E. Dawson and Martin Alexander, 1968. 2,4-D metabolism. Enzymatic degradation of chlorocatechols. Journal of Agricultural and food chemistry, vol. 16, pp. 829-833.

116. Cochrane, D. R., J. D. Pope, Jr., H. P. Nicholson, and G. W. Bailey, 1967. The persistence of silvex in water and hydrosoil. Water resources research, vol. 3, pp. 517-523.

Section IV

Efficacy Data Under Field and Laboratory Conditions

A. Effectiveness for Intended Purpose When Used as Directed:

Three decades of intensive research has shown that 2,4-D and 2,4,5-T are still our two most useful herbicides for controlling undesirable woody plants on forest lands. Both chemicals are of approximately equal value due to variation in susceptibility of our native shrubs and tree species. Silvex (2,4,5-TP) is not generally as effective as either 2,4-D or 2,4,5-T, and much smaller amounts of Silvex are used on forest lands. Many species, like vine maple (Acer circinatum) in the Pacific Northwest respond only to 2,4,5-T. These three herbicides are by far the most extensively used chemicals for woody plant control on forest land throughout the Nation. After 30 years of research, phenoxy herbicides are not only the most effective herbicides on woody plants on forest lands, but also the least expensive, the least persistent, and the most selective for control of undesirable woody species on forest land.

Substituting less effective herbicides would only increase contamination of the forest environment. Increased amounts of herbicide and additional applications of these herbicides would be required to achieve similar silvicultural results. It should also be noted at this time that the phenoxy herbicides 2,4-D, 2,4,5-T, and 2,4,5-TP are biodegradable and do not persist in the forest environment. Although this will be

stressed later, it should be noted at this time that by replacing these compounds, we may inadvertently select and use chemicals that are more damaging and persistent in the environment. We now have 20 years of accumulated knowledge concerning effects of 2,4,5-T on forest vegetation without visible evidence of adverse effect on humans, desirable vegetation, or wildlife. It is readily conceivable that new chemicals may prove even less acceptable after a similar period of use and study.

B. Phytotoxicity: (See tables)

It would be literally impossible to list the relative effectiveness of 2,4-D, Silvex, and 2,4,5-T on the multitude of plants treated with these herbicides in all parts of the Nation. Furthermore, the chemicals are applied as aerial sprays, basal sprays, stem sprays, and with powered ground apparatus in many different carriers. As stated earlier, there are also a host of different types of formulations available as commercial products containing acids, esters, amines, inorganic salts, etc. Therefore, the following pages simply show the relative effectiveness of low volatile esters of these three phenoxy herbicides when applied as aerial budbreak or foliar sprays. In general, their relative effectiveness when used in this fashion, is indicative of their relative effectiveness on the same species when applied in other ways. Although the list of species contains only a small percentage of those plants treated with 2,4-D, Silvex, and 2,4,5-T, the fact that this lengthy list is so incomplete

serves to stress the relative importance of the three chemicals in forests, on rights-of-way, range lands and pasture lands. If their safe agricultural uses were also listed, the size of the document would be overwhelming.

Table 1 RELATIVE EFFECTIVENESS OF 2,4-D, SILVEX, AND 2,4,5-T APPLIED AS AERIAL BUD-BREAK OR FOLIAR SPRAYS 1/

Problem Area 2/	Plant	Chemical 3/	Pounds ai per acre 4/	Diluent 5/	Season 6/	Results 7/	References
Woody Plants On	Acer circinatum	2,4,5-T	2	Oil	BB	I	18
Forest Lands	Acer macrophyllum	Silvex	4	Emulsion	EF	I	2
	Acer negundo	2,4-D	2	Water	LF	S	Melander, 1950
	Alnus spp.	2,4-D	2	Oil	BB	S	22
	Alnus rubra	BK	2	Oil	BB	S	18, 2, 14
		2,4-D	2	Emulsion	LF	S	21, 4
		2,4,5-T	2	Oil	BB	S	17
	Amelanchier alnifolia	2,4-D	1	Water	EF	S	14, 16
	Arbutus menziesii	2,4-D	2	Emulsion	EF	I	17
	Arctostaphylos spp.	2,4-D	3	Emulsion	EF	S	2, 18
	Arctostaphylos canescens	2,4-D	1	Water	EF	S	16
	Arctostaphylos columbiana	2,4-D	1	Water	EF	S	16
	Arctostaphylos hispidula	2,4-D	2	Emulsion	EF	S	16
	Arctostaphylos parryana	2,4-D	1	Water	EF	S	11
	Arctostaphylos patula	2,4-D	2	Water	EF	I	16
	Betula spp.	2,4,5-T	3	Emulsion	EF	S	7, 25
	Betula populifolia	2,4-D	1	Water	LF	I	Melander, 1950

Problem Area 2/	Plant	Chemical3/	Pounds ai per acre4/	Diluent5/	Season6/	Results7/	References
Woody Plants on Forest Lands (Continued)	Castanopsis chrysophylla	2,4,5-T	2	Emulsion	EF	I	16
	Castanopsis chrysophylla var. minor	2,4,5-T	2	Emulsion	EF	I	16
	Castanopsis sempervirens	2,4,5-T	1	Emulsion	EF	I	11
	Ceanothus spp.	2,4,5-T	3	Emulsion	EF	S	16, 17
	Ceanothus dordulatus	2,4,5-T	2	Water	EF	I	16
	Ceanothus integerrimus	2,4-D	2	Water	EF	S	16
	Ceanothus thyrsiflorus	2,4,5-T	3	Emulsion	EF	S	16, 17
	Ceanothus velutinus	2,4,5-T	2	Emulsion	EF	I	11, 16
	Ceanothus velutinus var. laevigatus	2,4,5-T	4	Emulsion	EF	S	16
	Cornus spp.	2,4-D	2	Water	LF	S	23
	Corylus spp.	BK	2	Water	EF	I	2, 23
	Corylus americana	2,4-D	1	Water	LF	S	Melander 1950
	Crataegus spp.	2,4,5-T	3	Water	EF	S	23
	Crataegus spp.	BK	2	Water	LF	S	Melander, 1950

1/ See footnotes at end of tables.

Problem Area 2/	Plant	Chemical3/	Pounds ai per acre4/	Diluent5/	Season6/	Results7/	References
Woody Plants On	Cytisus scoparius	2,4-D	2	Emulsion	EF	S	2, 17
Forest Lands	Garrya spp.	BK	4	Emulsion	EF	I	17
(Continued)	Gaultheria shallon 2,4,5-T		4	Emulsion	EF	R	2
	Hamamelis virginiana	BK	3	Water	LF	I	Melander, 1950
	Liquidambar styraciflua	2,4,5-T	3	Emulsion	EF	S	9
	Lithocarpus densiflorus	2,4,5-T	2	Emulsion	EF	I	16, 18
	Myrica cerifera	2,4,5-T	2	Water	LF	I	Melander, 1950
	(Northern hardwoods)	2,4,5-T	8	Oil	EF(Frill)	S	20
	Ostrya spp.	2,4,5-T	4	Oil	(cut surface)	S	23
	Pinus caribaea	2,4,5-T	2	Emulsion	LF	I	Amchem Prod. Inc
	Pinus echinata	2,4,5-T	2	Emulsion	LF	I	Amchem Prod. Inc
	Pinus lambertiana	2,4-D	2	Water	LS	R	13
	Pinus lambertiana	2,4,5-T	2	Water	LS	R	13, 8
	Pinus palustris	2,4,5-T	2	Emulsion	LF	I	Amchem Prod. Inc
	Pinus ponderosa	2,4-D	2	Water	LS	R	13
	Pinus ponderosa	2,4,5-T	2	Water	LS	R	13, 8
	Pinus Taeda	2,4,5-T	2	Emulsion	LF	I	Amchem Prod. Inc

Problem Area 2/	Plant	Chemical 3/	Pounds ai per acre 4/	Diluent 5/	Season 6/	Results 7/	References
Woody Plants	Populus spp.	2,4-D	3	Emulsion	EF	I	2, 17
On Forest Lands	Populus tremuloides	BK	2	Water	EF	S	23
(Continued)	Prunus spp.	2,4,5-T	4	Emulsion	EF	S	17, 24
	Prunus emarginata	BK	2	Emulsion	EF	I	---
	Prunus virginiana	2,4,5-T	3	Emulsion	LF	S-I	Est.
	Pseudotsuga menziesii	2,4-D	2	Emulsion	BB	R	13
	Pseudotsuga menziesii	2,4,5-T	2	Emulsion	BB	R	13
	Quercus chrysolepis	2,4-D	2	Emulsion	EF	R	16, 18
	Quercus garryana	2,4-D amine	50	Water	(cut surface)	S	12
	Quercus kelloggii	2,4,5-T	4	Emulsion	EF	I	17, 24
	Quercus marylandica	2,4,5-T	--	--	--	I	12
	Quercus minor	2,4,5-T	3	Emulsion	EF	I	12
	Rhamnus californica	2,4-D	4	Emulsion	LF	R	2
	Rhamnus purshiana	BK	4	Water	EF	I	2

1/ See footnotes at end of tables.

Problem Area 2/	Plant	Chemical 3/	Pounds ai per acre 4/	Diluent 5/	Season 6/	Results 7/	References
Woody Plants On	Rhododendron macrophyllum	2,4,5-T	16	Oil	Basal	R	2,13
Forest Lands	Rhus spp.	2,4,5-T	--	--	--	S	12
(Continued)	Rhus diversiloba	2,4,5-T	3	Emulsion	LF	I	2, 17
	Rhus hirta	2,4-D	2	Water	EF	S	23, 12
	Rhus typhina	2,4-D	2	Water	EF	S	Melander, 1950
	Ribes spp.	2,4,5-T	3	Emulsion	EF	I	17, 24
	Rosa spp.	2,4,5-T	2	Water	LF	S	23
	Rubus spp.	2,4,5-T	4	Emulsion	LF	I	17, 4
	Rubus parviflorus	2,4,5-T	3	Emulsion	EF	R	2
	Rubus spectabilis	2,4,5-T	4	Water	EF	S	18
	Salix spp.	2,4-D	2	Emulsion	EF	I	2, 4
	Salix nigra	BK	2	Water	LF	S	Melander, 1950
	Sambucus spp.	BK	2	Emulsion	EF	I	2, 17
	Sassafras	2, 4-D	--	--	--	S	12
	Symphoricarpos spp.	2,4,5-T	2	Water	LF	S	4
	Symphoricarpos alba	2,4-D	2	Water	EF	S	Melander, 1950
	Toxicodendron radicans	2,4,5-T	2	Water	EF	S	23

Problem Area 2/	Plant	Chemical3/	Pounds ai per acre4/	Diluent5/	Season6/	Results7/	References
	Ulex europeaus	2,4,5-T	3	Emulsion	EF	S	2, 4, 17
	Vaccinium spp.	2,4-D	4	Water	EF	S	23
	Zanthoxylum americanum	2,4-D	2	emulsion	EF	I	Melander, 1950
Herbaceous Plants	Ambrosia artemisifolia	2,4-D	2	Water	EF	S	2
Forest Lands	Centaurea solstitialis	2,4-D	1	Water	EF	S	2
	Cirsium arvense	2,4-D	4 aehg	Water	BB	I	2, 4
	Convolvulus arvensis	2,4-D amine	3	Water	EF	I	2
	Juncus effusus	2,4-D	4 aehg	Emulsion	EF	S	2
	Rumex spp. (not sheep-sorrel)	2,4-D	2 aehg	Water	EF	S	2
	Senecio jacobaea	2,4-D	4	Water	EF	S	2, 4
	Tribulus terrestris (Seedling stage)	2,4-D	10 aehg	Water	EF	S	2
	Typha latifolia	2,4-D	6 aehg	Emulsion	BB	S	2

1/ See footnotes at end of tables.

TABLE I - RELATIVE EFFECTIVENESS OF 2,4-D, SILVEX AND 2,4,5-T APPLIED AS AERIAL BUD-BREAK OR FOLIAR SPRAYS

Problem Area	Plant	Chemical	Pounds A.I. Per Acre	Diluent	Season	Results	Reference
Woody Plants on Rangeland	Adenostoma fasciculatum	BK	4	Emulsion	Spring	R	Chemical Control of Range Weeds, Chemical Plant Control Sub Comm. Range Seeding Equ ment Comm.; USDA USDI
	Artemisia cana	2,4-D	2	Emulsion	New twig growth 3"-4"	S	" " "
	Artemisia frigida	2,4-D	2	Emulsion	Spring	S	" " "
	Artemisia tridentata	2,4-D	2	Emulsion	New twig growth 3"-4"	S	" " "
	Cholla cacti	2,4,5-T	4	Emulsion	Jul.-Aug.	I	" " "
	Chrysothamnus nauseosus	2,4-D	3	Emulsion	New twig growth 3"	I	" " "
	Chrysothamnus viscidiflorus	2,4-D	3	Emulsion	" "	I	" " "
	Juniperus (spp.)	-	-	-	-	Phenoxys not effective	" "
	Larrea tridentata	-	-	-	-	" "	" "
	Opuntia platiyopuntia	2,4,5-T 2,4,5-TP	8-10	Emulsion	Jul.-Aug.	I	" " "
	Pinyons - Pinus edulis; Pinus monophylla; Pinus cembroides	-	-	-	-	Phenoxys not effective	" "

TABLE I - RELATIVE EFFECTIVENESS OF 2,4-D, SILVEX AND 2,4,5-T APPLIED AS AERIAL BUD-BREAK OR FOLIAR SPRAYS (Cont.)

Problem Area	Plant	Chemical	Pounds A.I. Per Acre	Diluent	Season	Results	Reference
Woody Plants on Rangeland (cont.)	Prosopis juliflora	2,4,5-T	1/2	Emulsion	40-90 days after first leaves appear	I	Chemical Control of Range Weeds, Chemical Plant Control Sub Comm.; Range Seeding Equipment Comm.; USI and USDI
	Quercus douglasii	2,4,5-T	4#/gal. cut surface	Water	Late winter	I	" " "
	Quercus stella	2,4,5-T 2,4,5-TP	2	Emulsion	Active growth after leaf maturity	R	" " "
	Quercus turbinella	BK	4	Emulsion	May	R	" " "
	Quercus dumosa	BK	4	Emulsion	May	R	" " "
	Quercus havardii	2,4,5-TP	1/2	Emulsion	May-June	I	" " "
	Salvia aithiopis	2,4-D	2	Water	Spring	I	" " "
	Sarcobatus vermiculatus	2,4-D	2	Emulsion	When making rapid growth	I	" " "
	Yucca glauca	2,4,5-TP	1	Emulsion	Pre-bloom stage	S	" " "
Forbs on Rangeland	Astragalus spp.	2,4-D	2	Emulsion	Full bloom	I	" " "
	Centaurea picris	2,4-D	4	Water	Early bud stage	I	" " "
	Cicuta spp.	2,4-D	2	Wager	Early bud stage	I	" " "

TABLE I - RELATIVE EFFECTIVENESS OF 2,4-D, SILVEX AND 2,4,5-T APPLIED AS AERIAL BUD-BREAK OR FOLIAR SPRAYS (Cont.)

Problem Area	Plant	Chemical	Pounds A.I. Per Acre	Diluent	Season	Results	Reference
Forbs on Rangeland (Cont.)	Delphinium spp. (low)	2,4-D	1½	Water	Before flower stalks are 2" high	I	Chemical Control Range Weeds, Chemical Plant Control Sub Comm.; Range Seeding Equipment Comm.; USDA and USDI
	Delphinium occidentals (tall)	2,4,5-T 2,4,5-TP	4	Water	Pre-bud stage	I	" " "
	Gutierrezia microcephala	2,4-D	2	Emulsion	New twig growth 3"-4" long	S	" " "
	Gutierrezia sarothrae	2,4-D	2	Emulsion	" "	S	" " "
	Halogeton glomeratus	2,4-D	2	Water	Early post-emergence	I	" " "
	Helenium hoopesii	2,4-D	3	Water	Pre-bloom stage	I	" " "
	Hymenoxys adurata	2,4-D	1	Water	" "	S	" " "
	Hypericum perforatum	2,4-D	4-6	Water	Early summer plants 6" high	S	" " "
	Iris missouriensis	2,4-D	3	Emulsion	Blooming	I	" " "
	Lupine spp.	2,4-D	3	Emulsion	Pre-bloom stage	I	" " "
	Madia glomerata	2,4-D	1	Water	Reach 4 leaf stage	S	" " "

TABLE I - RELATIVE EFFECTIVENESS OF 2,4-D, SILVEX AND 2,4,5-T APPLIED AS AERIAL BUD-BREAK OR FOLIAR SPRAYS (Cont.)

Problem Area	Plant	Chemical	Pounds A.I. Per Acre	Diluent	Season	Results	Reference
Forbs on Rangeland (Cont.)	Senecio jacobaea	2,4-D	2	Water	At rosette stage	S	Chemical Control of Range Weeds, Chemical Plant Control Sub Comm.; Range Seeding Equipment Comm.; USDA and USDI
	Veratrum californicum	2,4-D	3	Water	Before bloom	I	" " "
	Wyethia amplexicaulis	2,4-D	2	Water	Half bloom stage	S	" " "
	Wyethia helianthoides	2,4-D	2	Water	Half bloom stage	S	" " "
	Zigadenus venenosus	2,4 -D	2	Water	3-6 leaf stage	S	" " "

Problem Area 2/	Plant	Chemical 3/	Pounds ai per acre 4/	Diluent 5/	Season 6/	Results 7/	References
Herbaceous Plants On Rights-of-Way and on Industrial Sites	Broadleaf weeds in general	2,4-D	4	Water	EF	S	2
Basal and Cut Surface Treatments on Noncrop Land	NOTE: BASAL SPRAYS: low volatile esters are applied in oil carriers at rates of 8 to 16 aehg, spraying all stems to point of runoff up to 12 or 18 inches above ground. Best on thin barked species; thick barked trees are usually best treated by cut surface methods.						
	CUT SURFACE TREATMENTS: These include partial and complete frills, injector treatments, and hack-squirt treatments. Water soluble amines are usually most effective, but for some species and areas esters in oil are preferred. The amines are usually applied undiluted or diluted in 1:1 mixtures. Only one or two milliliters need be applied per cut.						

Footnotes for Table 1

2/ Apply low volatile esters unless otherwise specified.

3/ BK refers to brushkiller mixtures of 2,4-D and 2,4,5-T

4/ Pounds acid equivalent per acre except aehg which means pounds acid equivalent per hundred gallons of spray solution to be applied to drip point on foliage in spot treatments.

5/ Emulsions usually contain 1/2 to 1 gallon of No. 2 diesel oil per acre; diesel oil is also usually used as an oil carrier alone.

6/ BB = at time of bud break on the weed species; EF = early foliar, after about three-fourths of the leaves on deciduous species have reached full size and hardened; LF sprays are applied after all leaves have reached full size and terminal growth has ceased; LS sprays are applied in late summer (usually late August or very early September), but at least one month before leaf abscission on deciduous species.

7/ S = susceptible, usually controlled with one treatment.

I = intermediate in susceptibility; usually requires at least 2 treatments.

R = resistant; usually requires more than two treatments and may still show some sprouting and recovery.

C. Translocation with plant treated:

Translocation of phenoxy herbicides is closely associated with movement of organic foods from regions where sugars are synthesized to regions where food is being utilized in growth and storage (28, 33, and 36). Foliar applied herbicides move through the leaf cuticle into the living cells and are then rapidly translocated through living tissues (symplast) such as the lumina of sieve tubes in the phloem (29, 34). Translocation is to areas where food is being used or stored at the moment. For example, from leaves of young seedlings, movement is into the roots, resulting in excellent plant kill. From later leaves, foods and herbicides are moved into both roots and shoots; giving some of the best kills on older plants. Movement from mature upper leaves is largely into growing shoots, flowers, and fruits (29). In this season, herbicides may begin to decrease somewhat in effectiveness.

In the process, appreciable amounts of the herbicide may be lost. Some of this is absorbed on plant surfaces, other portions may not be able to penetrate the plant cuticle and are leached from the surface by rain, some of that which is absorbed into the plant may be immobilized in the treated tissue by absorption to or conjugation with cell constituents, additional portions may accumulate in vacuoles of parenchyma, and finally, additional portions may be degraded by enzyme systems within the plant.

Under any circumstances, movement of phenoxy herbicides in woody plants varies considerably depending upon species, season, and formulation of herbicide. Leonard and Crafts (33) tested several herbicides on seven different species of shrubs in California. All species showed different patterns of upward and downward movement during various seasons of the year. Coyote bush absorbed and translocated radioactive 2,4-D only slightly in February, intensely in April, and not at all in July. In February and March, movement was downward from treated leaves; in April almost entirely upward. In manzanita, 2,4-D was absorbed and translocated throughout May, but most of the movement was downward. On live oak (Quercus wislizenii) 2,4-D was actively translocated from February through September: in February, movement was entirely downward, some upward after new growth started in March, but then largely downward throughout the rest of the season. Presence of adequate soil moisture for food manufacture and movement was important in activity of the herbicides as well. Their study also showed that too quick a leaf kill nullifies the effect of the herbicides. An early browning of leaves, reduces absorption and translocation of the herbicides and results in an ineffective treatment. This stresses the fact that different species require different treatments both in rate of herbicide and time of application. In evergreen species, chemicals may move up and down the stems for many months; the period of movement may be very short for deciduous species. Others (28) learned that translocation of phenoxy herbicides is much slower in woody seedlings

than in herbaceous seedlings (1) and that high humidity increases uptake by oak seedlings. Yamaguchi (37) learned that 2,4-D and probably the other phenoxy herbicides as well, translocate much less readily than amitrole. He also found that 2,4-D moves into plant leaves better from acidic solutions than from an alkaline medium. Approximately ten times as much, 2,4-D was absorbed from a medium of pH 3 then from one with a pH 11.

In basal sprays, low volatile esters dissolved in oil are heavily applied to the lower 18 inches of stems of shrubs and weed trees. Such treatments are usually most effective during the growing season, but are usually no better than foliar sprays on most species. In basal bark applications, the herbicides move upward through the xylem with the transpiration stream. From there, there is increasing evidence that such material may move into living tissues such as the phloem and be translocated to other parts of the plant. On thin-barked species, there is evidence that 2,4,5-T may be picked up as readily through the thin bark as through the foliage (34). In this experiment, 2,4,5-T was tested in three different formulations: an ester, an amine, and an acid. In all cases, the ester form was picked up best.

Two additional points deserve mention. Freed and Morris (30) have pointed out that ecotypic variation within species can account for successful effects on a species in one area, while the same treatment fails on the same species in another area.

This was first observed some years ago. The second point is that phenoxy compounds, like other herbicides, may leak from roots into soil, where they may affect soil microflora and microfauna. This could have some hidden and unforseen effects in silviculture. Such effects, however, may be counteracted to some extent by increased soil temperature and moisture that produce increased amounts and activity of soil microflora and microfauna (31).

D. Compatability

The phenoxy herbicides are compatible with each other and with most other herbicides, but this compatibility is dependent to a great degree on the particular formulation of 2,4-D silvex, or 2,4,5-T used. Phenoxy herbicides formulated for use only in oil carriers should never be mixed with wettable powders like atrazine or other materials formulated only for use in water carriers. Water soluble formulations such as amines or inorganic salts can be safely mixed with other water soluble herbicides or wettable powders. Most phenoxy esters, however, are formulated for use in either water, oil, or oil-in-water emulsions. Such formulations can be mixed and applied simultaneously with oil-soluble or water-soluble materials or with wettable powders such as atrazine, terbacil, and dalapon.

Phenoxy herbicides are also sometimes considered for mixture and application with fertilizers. When considered for simultaneous application with phenoxy herbicides, however, the fertilizer must be either water soluble or in liquid form. Recent research also indicates that ammonia and urea fertilizers have different effects on action of phenoxy herbicides. This should be considered before making a choice of fertilizers.

REFERENCES

1. 1972. Specimen labels useful as references for these herbicides may be obtained from:

   Amchem Products, Inc.

   Chipman Chemical Company, Inc.

   Diamond Alkali Company

   The Dow Chemical Company

   Monsanto Chemical Company

   Stauffer Chemical Company

   Thompson-Hayward Chemical Company

2. Oregon Extension Service. 1970. Oregon weed control handbook. Oregon Univ., Coop. Ext. Serv., Corvallis, Oregon 287 pp.
3. National Academy of Sciences, 1957. Principles of weed control, Vol. 2, Weed Control, 471 pp.
4. Washington State University and Department of Agriculture, 1971. Washington pest control handbook. Washington State Univ., Pullman, Washington, 569 pp.
5. Weed Society of America, 1967. Herbicide handbook of the Weed Society of America. W. F. Humphrey Press, Inc., Geneva, N.Y., 293 pp.
6. Anonymous, 1961. Herbicides and their use in forestry. Symp. Proc., School of Forestry, Oreg. State Univ., Corvallis, Oregon, 122 pp.
7. Arend, John L., and Eugene I. Roe, 1961. Releasing conifers in the Lake States with chemicals. USDA, Agric. Handbook No. 185, 22 pp.

8. Bentley, Jay R., and Kenneth M. Estes, 1965. Use of herbicides on timber plantations in California. California Region USFS and Pacific SW Forest and Range Experiment Station, USDA, 47 pp.

9. Brady, Homer, Fred A. Peev, and Paul Y. Burns, 1969. Erratic results from aerial spraying of midsouth hardwoods. Journal of Forestry 67(6): 393-396.

10. Burns, Paul Y. (Ed.), 1958. Chemical pine release symposium, Proc. The Dow Chemical Co., Louisiana State Univ. 67 pp.

11. Dahms, Walter G., 1961. Chemical control of brush in ponderosa pine forests of Central Oregon. US Forest Service, Res. Pap. 39, 17 pp, Pacific Northwest Forest and Range Exp. Sta., Portland, Oregon.

12. Gratkowski, H., 1959. Effects of herbicides on some important brush species in southwestern Oregon. U.S. Forest Service Res. Pap. 31, 33 pp., Pacific Northwest Forest and Range Experiment Station, Portland, Oregon.

13. 1961, Toxicity of herbicides on three northwestern conifers. U.S. Forest Service Res. Pap. 42. 24 pp., Pacific Northwest Forest and Range Experiment Station, Portland, Oregon.

14. 1961. Use of herbicides on forest lands in southwestern Oregon. U.S. Forest Serv. Res. Note No. 217. 18 pp. Pacific Northwest Forest and Range Experiment Station, Portland, Oregon.

15. Gratkowski, H. J. and J. R. Philbrick, 1965. Repeated aerial spraying and burning to control sclerophyllous brush, Jour. Forestry, 63(12):919-923.

16. Gratkowski, H., 1968. Repeated spraying to control southwest Oregon brush species. U.S. Forest Serv. Res. Pap. PNW-59. 6 pp. Pacific Northwest Forest and Range Experiment Station, Portland, Oregon.

17. Leonard, O. A. (Ed.) 1961. Tables on reaction of woody plants to herbicides. Western Weed Control Conf. Res. Prog. Rpt. pp. 27-37.

18. Leonard, Oliver A., and W. A. Harvey, 1965. Chemical control of woody plants. Calif. Agric. Exp. Sta., Davis, Calif. Bull. 812, 26 pp.

19. Lindmark, Ronald D., 1965. Removing undesirable trees from hardwood stands. Central States Forest Experiment Station, U.S. Forest Service, USDA, 23 pp.

20. McQuilkin, W. E., 1957. Frill treatment with 2,4,5-T and 2,4-D effective for killing northern hardwoods. Northeastern For. Exp. Sta., Station Paper No. 97, 18 pp.

21. Newton, Michael (Ed.) 1967. Vegetation management--system of operation. Symp. Proc.: Herbicides and Vegetation Management in Forests, Ranges, and Noncrop Lands. Oregon State University, Corvallis, Oregon. pp. 8-11.

22. Romancier, Robert M., 1965. 2,4-D, 2,4,5-T, and related chemicals for woody plant control in the southeastern United States. Georgia Forest Research Council Report No. 16. 46 pp. Southeastern Forest Exp. Sta., US Forest Service, USDA.

23. Rudolf, Paul O., and Richard F. Watt, 1956. Chemical control of brush and trees in the Lake States. Station Paper No. 41, 58 pps. Lake States Forest Exp. Sta., US Forest Service, USDA.

24. Schubert, Gilbert H., 1962. Chemicals for brush control in California reforestation. US Forest Service Misc. Pap. 73, 14 pp. Pacific Southwest Forest and Range Experiment Station, USDA.

25. Sutton, R. F., 1958. Chemical herbicides and their uses in the silviculture of forests of eastern Canada. Forest Research Branch Technical Note No. 68, 56 pp. Canada Dept. of Agri.

26. Tschirley, Fred H. (Ed.) 1968. Research report...response of tropical and subtropical woody plants to chemical treatments. Dept. of Agric. CR-13-67, 197 pp. Agric. Res. Serv., Washington, D. C.

27. Walker, Laurence C., 1956. Controlling undesirable hardwoods. Georgia Forest Research Council Rep. No. 3, 24 pp. School of Forestry, University of Georgia, Athens, Georgia.

28. Clor, M. A., A. S. Crafts, and S. Yamaguchi, 1964. Translocation of C^{14}-labeled compounds in cotton and oaks. Weeds 12(3): 194-200.

29. Crafts, A. S., 1961. The chemistry and mode of action of herbicides. Interscience Publ. N.Y., 269 pp.

30. Freed, V. H., and R. O. Morris, 1967. Environmental and other factors in the response of plants to herbicides. Agric. Experiment Sta. Tech. Bull. 100. 128 pp, Oregon State University, Corvallis, Oregon.

31. Gratkowski, H., 1967. Ecological considerations in brush control. In herbicides and vegetation management, Symp. Proc., Oregon State Univ., Corvallis, Oregon, pp. 124-140.

32. Leonard, O. A., 1963. Translocation of herbicides in woody plants. Soc. of American Foresters Proc.

33. Leonard, O. A., and Alden S. Crafts, 1956. Uptake and distribution of radioactive 2,4-D by brush species. Calif. Agric. Exp. Sta. University of California, Berkeley, California, Hilgardia 26(6): 366-415.

34. Leonard, O. A., D. E. Bayer, and R. K. Glenn, 1966. Translocation of herbicides and assimilates in red maple and white ash. Bot. Gaz. 127(4):193-201.

35. Shaw, W. C., J. L. Hilton, D. E. Moreland, and L. L. Jansen, 1960. Herbicides in plants. Symp. Proc., The nature and fate of chemicals applied to soils, plants, and animals. pp. 119-133. Agric. Res. Serv., U.S. Dept. Agric. Beltsville, Maryland.

36. Woods, Frank W., 1955. Control of woody weeds. U.S. Forest Serv. Occ. Pap. 143. 50 pp., Southern Forest Experiment Station, New Orleans, Louisiana.

37. Yamaguchi, Shogo, 1965. Analysis of 2,4-D transport. Div. of Agric. Sci., Univ. of California, Berkeley, California, Hilgardia 36(9):349-377.

Section V Residues

A. Residues in Soil

The forest floor is a major receptor of phenoxy herbicides whether applied from aircraft or by ground spray systems. There it may be absorbed on soil colloids or absorbed in organic matter, degraded chemically or biologically, volatilize and move to other areas, or leach to depths or locations where it cannot be absorbed by plant roots (11, 13). Once in the soil, however, the phenoxy herbicides are not persistent (9, 11, 13, 14). 2,4-D is much more rapidly degraded than 2,4,5-T or silvex, but even 2,4,5-T will not usually remain in the forest floor from one year to the next. Fairly rapid degradation of phenoxy herbicides in soil has been shown in several studies (9, 10, 11, and 14). Generally, these studies indicate that 85 to 90 percent of 2,4-D will be degraded in about 15 days, but 2,4,5-T is more persistent. In one study, 23 percent of 2,4,5-T was still present in soil after 13 days; this had decreased to 13 percent after 120 days.

The concensus is almost unanimous that degradation of phenoxy herbicides in soil is microbial (4, 5, 7, 11, 14, and 15). Steenson (16) believed that bacterial decomposition is aerobic. Norris' (11) data indicates that soil microorganisms adapt more readily to use 2,4-D than 2,4,5-T as a substrate, and that it is for this reason that 2,4-D is degraded more quickly than 2,4,5-T

and possibly silvex (7). Repeated applications are more rapidly decomposed by microbes than are the initial sprays (9, 10). This would indicate that once adapted to use these materials as a substrate soil microorganisms begin decomposition more quickly or their numbers increase more rapidly when phenoxy herbicides once again appear in their soil environment.

Finally, different formulations show different rates of degradation. Pure 2,4-D acid degraded more quickly than either the solubilized acid or the isooctyl ester (12). Both acid and ester forms are leachable from soil. Leaching and movement into streams does not usually become a problem however, since the herbicides are rapidly decomposed in the soil (10).

Recent studies show that 2,4,5-T is extensively adsorbed by forest floor material. About 60 percent of the 2,4,5-T in solution was adsorbed at equilibrium (30°C) which was attained in a few hours (14). The extensive interaction of 2,4,5-T with the forest floor suggests only limited leaching should occur. In an agricultural soil, 2,4,5-T remained in the upper six inches even after application of 4.5 inches of water over a short period of time (17). A lack of 2,4,5-T residues in streams flowing from treated areas (13) suggests that a combination of rapid degradation and resistance to leaching prevents stream contamination. (8)

Greenhouse tests using beans as the more, and tomatoes as the less, sensitive indicator crop on several typical agricultural soil types in Hawaii showed that 2,4-D applied as dust at 10 lb/acre was dissipated from the soil in 2-14 weeks, the rapidity of inactivation depending on the higher temperatures and pH values of the soil. A change of pH also was not correlated with organic-matter content, fertilizers or adsorption capacity. The number of aerobic bacteria appeared to be negatively correlated with 2,4-D persistence in the soil (1).

After leaching a 25 cm-high soil column to which 2,4-D had been applied superficially, almost the entire application was found in the top 5 cm. At recognized application rates, and under favorable conditions of moisture and temperature for soil microorganisms, 2,4-D was inactivated in a maximum period of six weeks. In a forest clearing on relatively "inactive" acid soil, a heavy application of 2,4-D was rendered 90% inactive in 15 weeks, but in sterilized soil no decomposition of the substance was observed. Its inhibiting action on germination was markedly stronger in sandy than in loamy soils. Nitrification in nutrient solutions was considerably checked at the normal application rate of 2,4-D, but addition of soil almost completely counteracted this effect. (3)

The presence of Tordon 50 D (picloram tri-isopropanolamine 2,4-D in the proportions 1:4) and 2,4,5-T in soil reduced the emergence

and survival of Pinus radiata seedlings, the major effect being on survival. Effects of 2,4,5-T disappeared rapidly (within two months of application), whereas some effects of Tordon 50 D persisted for at least six months. Sterilization of the soil by autoclaving did not delay breakdown of the herbicide, nor did leaching of the soil hasten the decline of its activity. The herbicides affected initial growth of seedlings, but older seedlings would suffer little or no damage if planted on sites treated with these herbicides a few months previously (2).

The repeated applications of 2,4-D to soils resulted in a buildup of organisms which rapidly decompose the hormone. Two bacterial species were isolated from such treated soils; these were demonstrated to be capable of inactivating 2,4-D added to the soil. These were identified as Flavovacterium aquatile and a Coryne-bacterium-like organism. Approximately ten times as much 2,4-D as dinitro-o-cresol was required to inhibit the growth of the following soil organisms: Rhizobium meliloti, R. trifoli, R. lebuminosarum, R. lupini, Agrobacterium radiobacter, Azotobacter chroococcum, A. beijerinckii, Nitrosomonas europaea, Bacillus subtilis, B. mycoides, Escherichia coli, Bacterium aerogenes, B. prodigiosum, Pseudomonas pyocyanea, Cellvibrio sp., Sytophaga sp., Mycobacterium phlei, Nocardia corallina, Streptomyces griseus, and Micromonospora sp. It is considered unlikely that the repeated applications of 2,4-D to soils would

seriously inhibit the beneficial bacterial flora. It is suggested that there is a possibility that the repeated applications might result in an excessive increase in the bacterial flora which would inactivate the hormone and thus result in a decrease in the effectiveness of the herbicide (6).

B. Residues in Water

All available information indicates that although some phenoxy herbicides may enter streams flowing through or adjacent to areas being sprayed, the levels in the streams will be very low. In 6 years of monitoring spray operations in western Oregon, scientists have never found phenoxy residues exceeding 0.1 ppm in western Oregon streams (29, 31). Even this can be reduced or eliminated by leaving untreated buffer strips between the sprayed area and running streams. Such short-term initial low-level contamination by 2,4,5-T is regarded as no hazard to fish or animals. Long-term low-level pollution is only found where phenoxy herbicides are applied directly on marshy areas.

In their report to Administrator W. D. Ruckelshaous of the Environmental Protection Agency, the Advisory Committee of Scientists on 2,4,5-T stated that all available data indicates that the amount of 2,4,5-T entering water is small and doesn't stay long. It is adsorbed on clay or absorbed by viota within

days (36). Phenoxy chemicals entering water may be lost by volatilization, adsorption on sediments, absorption by biota, by degradation, and by dilution as additional stream water passes through the site. This latter function is by far the most important. Almost all authorities agree that there is adsorption on bottom sediments (18, 22). This contamination of bottom sediments, however, does not appear to last long. Concentrations of low volatile esters of silvex in water after application on the surface of three ponds decreased to 0 by the end of three weeks (18). Rapid degradation of phenoxy herbicides in water appears to be the rule, especially in bodies of water with histories of repeated applications of phenoxy herbicides. Several studies indicate further that persistence of 2,4-D and 2,4,5-T in fresh water ponds can be drastically decreased by adding small amounts of soil previously treated with phenoxy herbicides (32). Rapid degradation of 2,4-D was also observed in water samples collected from areas with a history of repeated 2,4-D applications (23).

As stated earlier, most phenoxy herbicides enter aquatic environments during the actual period of spraying. There appears to be little chance that additional amounts will be added to the water with the passage of time. At normal application rates, approximately 100 to 300 ppm of herbicide will be found in vegetation shortly after application. This will decline to low levels in a few weeks (31).

Only small amounts of herbicide will enter streams by washing action of rain from overhanging treated vegetation above a stream or from leaves falling into water (29). Furthermore, repeated observation indicates that heavy fall rains will not leach phenoxy herbicides through the soil into streams if the herbicides have been applied during the spring or very early summer. The phenoxy herbicides move through the soil only in very small amounts and for very short distances. There seems very little chance of stream pollution from this source (30). Although small amounts of phenoxy herbicides are exuded from roots of treated plants, this also is a negligible source of contamination. The amounts exuded are small and only roots in the stream or in the hydrosoil would provide a source for such contamination (29). Such exhudation seems to occur most from plants that are photosynthesizing most rapidly (25).

In conclusion, it appears that it would be quite safe to continue use of the phenoxy herbicides on forest lands. Where these chemicals are used at rates up to 4 lbs. ae per acre and are properly applied, there should be little or no danger to aquatic environments in the treated areas or to nontarget organisms on the sprayed sites.

Since October 1965, samples of a water-suspended sediment mixture from 11 streams in the Western United States have been analyzed monthly for 12 pesticides including the herbicides 2,4-D, 2,4,5-T, and silvex. No herbicide was found at any station during the first year of the sampling program.(19)

Concentrations of (2,4-dichlorophenoxy) acetic acid (2,4-D) were determined in irrigation water following bank applications for weed control. Applications of 1.9 to 3 lb/A of 2,4-D produced maximum concentrations of 25 to 61 ppb. Reduction of herbicide levels appeared to be due to dilution as the water flowed downstream. Rates of reduction in herbicide levels showed that negligible concentrations would remain after the water traveled a distance of 20 to 25 miles. The low concentrations of herbicides observed in the irrigation water likely would not be hazardous to crops or animals.(21)

In studies of the persistence of silvex in a closed artificial aquatic environment in the laboratory, mean concentrations ranged from 820 ppb immediately after application to 46 ppb after 19 weeks (Cochrane et al., 1965)(21). The ester of silvex was rapidly hydrolyzed to the free acid. The authors speculated that, in addition to loss through adsorption on hydrosoil or absorption by aquatic vegetation, degradation also occurred (21).

A field study of silvex persistence was carried out in a creek having very little water movement (Cochrane et al, 1965 (21) and Nicholson, unpublished data). After the first application, concentrations ranged from 83 ppb one day after spraying to 1.1 ppb after 21 days. Silvex was not detectable after 35 days. After the second application, concentrations ranged from 19 ppb one day

after spraying to 0.48 ppb after 70 days. Following the third application, concentrations decreased from 73 ppb immediately after spraying to 2 ppb after 48 days, and to 0.1 ppb after 6 months. Silvex could not be detected one year after application. Another field test was carried out in a fast-flowing stream which provided maximal opportunity for dilution and interchange of water. Residues of silvex were not detected except during the first few hours following treatment. The maximum level found in this study was 0.05 P.P.M (21).

C. Residues in Plants

Few studies have been conducted on herbicide residues in woody plants, and even some of this limited information is questionable or contradictory. Even less work has been done on the multitude of possible metabolites and their incorporation into or conjugation with plant constituents.

Persistence of 2,4-D, silvex, and 2,4,5-T in plants, is initially dependent upon the amount of herbicide that actually reaches the plant surface, and the percentage of this herbicide that is absorbed by the plant. In aerial application, the percentage reaching vegetation may be small. Interception disks at vegetation level on one area in the Oregon Coast Range indicated that only about one third of the herbicide applied was reaching the vegetation (48). Once within the plant, the herbicides are rapidly absorbed into the symplast and moved through the vascular channels along

with assimilates toward sinks where foods are being used. Enroute their concentration may be further reduced by accumulation in vacuoles of living parenchuma cells of phloem, cortex, xylem, and pith (39). Additional amounts may be metabolized through degradation of the acetic acid side chain, hydroxylation of the aromatic ring, or conjugation with a plant constituent (44). Some herbicide may even be immobilized by adsorption to cells or cell constituents at any point along this route (50). As a result, because of degradation, growth dilution, and other factors, residues of the phenoxy herbicide in plants will probably be markedly reduced within a few weeks after application (47).

A review of the literature indicates the concensus that phenoxy herbicides are among the least persistent herbicides in plants. Most investigations indicate metabolism of 2,4-D and 2,4,5-T in plants is similar, although their rate of degradation may vary considerably even within a given species or genus. Metabolism is much slower in dormant than in active tissues. Basler et al (38) found that excised blackjack oak leaves broke down 59% of 2,4,5-T into three major unidentified metabolites in 24 hours, and Morton (45) reported that 80% of 2,4,5-T applied to mesquite was metabolized in 24 hours. The environment of mesquite prior to treatment also affects the amount of phenoxyacetic herbicides metabolized. 2,4,5-T metabolism was greatest in the range from 70° to 8 °F., less at 100°F., and completely inhibited at 50°F. In

another study, Morton et al (46 found that initial concentrations of 100 ppm of 2,4-D or 2,4,5-T in grasses were decreased to 1 ppm and 2 ppm, respectively, after 16 weeks. They concluded that the half life of 2,4,5-T esters in green grass tissues ranged from 1.6 to 2.9 weeks.

Only a small percentage of the numerous metabolites of the phenoxy herbicides in plants have been identified. In sweet gum and southern red oak, 2,4,5-trichlorophenol was identified as a common metabolic product (40). It is conceivable that other metabolic by-products of the herbicides are utilized as constituents of the numerous carbohydrates, amino acids, and the numerous proteins in plants.

In their report to the Administrator of the Environmental Protection Agency, Wilson et al (52) concluded that 2,4,5-T doesn't accumulate in plants or in any other compartment of the biosphere and that risk of human exposure to 2,4,5-T in food, air, or water is negligible. They stated that in 10,000 food and feed samples from 1964 through 1969, only 25 contained trace amounts of 2,4,5-T (less than 0.1 ppm).

As Norris (49) points out, these reports and the extensive healthy resprouting of brush which commonly occur a year following spraying on forest lands, suggests that high residues of 2,4-D, silvex, and 2,4,5-T do not persist for long periods in forest vegetation. Since degradation processes in both soil and vegetation are quite similar, and the phenoxy herbicides do not persist from year to year in soil, it is also improbable that they would persist from one year to another in vegetation.

Other mechanisms also affect herbicides that are intercepted by foliage. Herbicides adsorbed on the surface of leaves will be washed off by winter rains, subjected to photodecomposition, and degraded by microbes. That portion that is leached from the surface will enter the forest floor and be degraded as described earlier. It has been determined that bacterial degradation products of phenoxy herbicides are carbon dioxide, inorganic chloride ions, and water. Since all of these materials are normal parts of our environment, such decomposition products are readily recycled and used by forest vegetation.

Much of the furor concerning teratogenic effects of 2,4,5-T centered on its contamination with TCDD (2,3,7,8-tetrachloro-dibenzo-p-dioxin). A slight change in the manufacturing process and strict quality control now ensure that commercial herbicidal products contain less than 0.1 ppm of TCDD--a level that poses no hazard when the products are used at recommended rates.

Recent studies by scientists of the U.S. Department of Agriculture should ease the minds of those concerned about possible effects of TCDD in the environment (42). Their research shows that TCDD is not biosynthesized from chlorophenols, in soils, is not a photo product of 2,4,5-trichlorophenol, and does not leach into the soil profile. Further, TCDD is not absorbed into or translocated within

the plant from foliar applications, and is not taken up by plants from the minute residues that might be present in soils. To be absolutely sure, Kearney and his associates treated the soil in this experiment with a concentration of TCDD approximately 40,000 times greater than the amount that would be deposited in soil from a 2-pound-per-acre application of 2,4,5-T contaminated with 1 ppm of TCDD. This was incorporated in the top 1/3-inch of the soil surface. About half of the TCDD applied to foliage could be washed from the leaves by simulated rainfall 2 hours after application.

Finally, TCDD could not be detected even at a level of 0.5 ppm in tissue extracts from 22 bald eagle carcasses. The scientists concluded that contamination by chlorodioxin in 2,4,5-T has produced no measurable effects on the environment.

It is evident from the way 2,4,5-T is used and its behavior in the forest environment that the primary exposure of animals to this chemical will be through consumption of treated vegetation. Let us consider the amounts of 2,4,5-T which might be ingested from the highest residues found (300 ppm) in the study by Morton et al. (1967) (47). A high milk-producing animal might consume up to 10 percent of its body weight in green forage per day. A 1,000 pound animal consuming 100 pounds of forage containing 300 ppm of 2,4,5-T would ingest 30 milligrams of 2,4,5-T per kilogram of body weight, well below the toxic level.

This is a maximum exposure and would be received only when ingesting forage grasses shortly after treatment. If residue levels drop to less than 10 ppm a few weeks after treatment (Morton et al. 1967)(47), the ingestion level of 2,4,5-T will be no more than 1 mg/kg.

Low-volatile and high-volatile esters of 2,4-dichloro-phenoxyacetic acid (2,4-D) were sprayed on separate pastures at about double the usual rate (43). Milk from cows grazing these pastures contained from 0.01 to 0.09 ppm 2,4-D during the first 2 days after spraying and lower amounts thereafter. Residues in milk from cows put into the pastures 4 days after spraying were below 0.01 ppm, the practical limit of precision of the method used. Residues of 2,4-D, in or on forage, declined rapidly during the experiment. Almost all the 2,4-D in or on forage was hydrolyzed to the acid form in samples of forage taken within one-half hour after spraying with the butyl ester of 2,4-D, and about 75% after applying the 2-ethylhexyl ester.

The herbicides 2,4-dichlorophenoxyacetic acid (2,4-D) and 2,4,5-trichlorophenoxyacetic acid (2,4,5-T), each labeled in the carboxyl position were sprayed on a pasture consisting of a mixture of silver beardgrass (Andropogon saccharoides Swartz.), little bluestem (A. scoparius Michx.), and dallisgrass (Paspalum dilatatum Poir.) and a sideoats grama (Bouteloua curtipendula (Michx. Torr)) pasture over a 3-year period (47). Plant samples were harvested at intervals between 1 hour and 16 weeks after treatment and residues determined

by radio assay. No important differences were found in the persistence of herbicides or of different formulations of the same herbicide. Rainfall was the most important factor influencing the persistence of the herbicides. The little bluestem-silver beardgrass-dallisgrass samples harvested 1 hour after treatment with the butoxyethyl ester of 2,4,5-T contained both this ester and the acid of 2,4,5-T. One week after treatment, the acid of 2,4,5-T and unknown metabolites were found but no ester (47).

Two chemicals were tested. One, an ester of 2,4,5-T, was considered a representative formulation of the commercially available herbicides. The other was 2,4,5-T in the form of an emulsifiable acid (51). Herbicides were applied to all streambank vegetation by the same operator. Since there was interest in detecting maximum contamination, the herbicides were applied during a low streamflow period. Flow in all streams was less than 0.1 cubic foot per second (45 gpm).

Water samples were taken periodically after treatment at various locations up and down stream. The first samples were collected immediately after spraying following by a second group of samples four hours later. Thereafter, samples were collected daily during the first week and twice a week during the next three weeks. Additional samples were collected after each rainstorm.

Streamflow samples collected were tested for contamination by a calibrated three-member odor panel (Figure 2). The testing procedure used was that approved by the American Society for Testing and Materials Results indicate that during the three weeks following treatment, contamination of streamflow occurred only immediately after spraying and after the first large storm. In addition, contamination was detectable only within the treated reach of stream and no contamination was ever found in a downstream sample. Downstream samples were collected approximately one mile away from the treated areas and in both locations below the junction of the two treated streams.

D. Residues in Air

A certain portion of the spray material is dispersed by the wind as fine droplets. Additional amounts of chemical may be lost through volatilization of spray materials falling through the air or from intercepting surfaces. Most of the herbicide not lost through drift or volatilization is intercepted by the vegetation or the forest floor. Additional small amounts fall directly on surface water (56).

The actual amount of "drift" and volatilization is dependent on a number of physical, chemical and environmental factors, some of which can be controlled or avoided by the applicator. Among the physical variables are the pressure of application, height and

speed of delivery, nozzle design and the like. Chemical factors include the properties of the carrier and the herbicide. Environmental factors include temperature, relative humidity and wind (56).

Losses of aerially applied chemicals by drift and volatilization should be avoided. First there is the obligation to hold environmental contamination to a minimum. Secondly, the more chemical which reaches the target the greater the efficiency of the operation (56).

The distribution of two pounds per acre 2,4,5-T applied as a mixture of low volatile esters in diesel oil was determined in the coast range near Eddyville, Oregon. Treatment was by fixed wing aircraft in the early spring. Analysis of interception disks show an approximate 60 percent to 75 percent loss of herbicide (57).

In a survey in the State of Washington, 2,4,5-T was detected 9 days out of 99 at Pullman, in average concentration in positive samples of 0.045 ug/m^3. At Kinnewick it was found 14 days out of 102 at average concentration in positive samples of 0.012 ug/m^3 (53). In Cincinnati, Ohio, 0.04 ppm was found adsorbed on dust in a trace of rain persumably from applications in Texas (55). Photochemical degradation would be expected to occur in the air, partic-

ularly at high altitudes and in dry climates where ultraviolet radiation is highest. Kearney et al (54) report that exposure of 5 and 10 ppm water solutions of 2,4,5-T to ultraviolet light from a 450 watt Hanovia lamp greatly reduced the 2,4,5-T present within 5 minutes. It is not possible to extrapolate accurately from these data to the rate of decomposition in sunlight, but it is obvious that photochemical degradation could play a significant role. Probably most of the 2,4,5-T that gets into the air very soon either settles out or is washed out by rain and thereby is returned to soil and water (58).

There is no evidence to suggest that 2,4,5-T remains in the air for more than a few weeks after insertion (58).

E. Residues in Animals

Feeding studies with various animals have shown that the phenoxy herbicides are rapidly excreted. Erne (1966) (60) reported the major route of elimination of 2,4,5-T from pigs, calves, and rats dosed with 100 mg/kg. was in the urine. Repeated doses did not result in retention or accumulation of herbicide. A cow which received 5 ppm 2,4,5-T in its feed eliminated essentially all of the chemical within two days following exposure, and no 2,4,5-T was found in the milk (62). Mice injected wih 100 mg/kg 2,4,5-T eliminated approximately 70 percent within 24 hours (63).

Evaluation of animal exposure to 2,4,5-T leads to the following conclusions: (26)

1. Dairy and beef animals allowed to forage on treated grasses will ingest highest concentrations of 2,4,5-T shortly after application.
2. Because of degradation, growth dilution, and other factors, residues of 2,4,5-T will be markedly reduced a few weeks after application.
3. The herbicide is rapidly excreted; there is no accumulation in animal tissues.
4. There is no detectable residue in milk; therefore, man will not be exposed to 2,4,5-T through consumption of milk or meat from animals foraging on treated grasses.
5. Long-term chronic exposure of wildlife should not occur since 2,4,5-T does not persist for long periods in the forest, and repeated applications are rare.

The distribution and elimination of two phenoxyacetic acids, 2,4-D and 2,4,5-T were studied with a chemical method in rats, pigs, calves and chickens (60).

When administered orally as amine or alkali salts, the compounds were readily absorbed and distributed over the organism in all species studied. The absorption of 2,4-D in the form of an ester

was incomplete, however, the ensuing plasma and tissue levels of 2,4-D being only low. (Intact ester could not be detected in plasma) (60)

The highest tissue levels of 2,4-D and 2,4,5-T were found in liver, kidney, lung and spleen, the levels sometimes exceeding the plasma level. In blood cells, 10-20% of the plasma level was found. Penetration of 2,4-D into adipose tissue and into the central nervous system was restricted, whereas a ready placental transfer was demonstrated in swine. The distribution pattern did not show any significant species or--in rats--sex differences. (60)

Elimination of the compounds was rapid, the plasma half-life being about three hours in rats, about eight hours in calves and chickens and about 12 hours in pigs. The tissue half-life values ranged between five and 30 hours, the lower values being found in rats. No retention in tissues was noted, nor was accumulation seen on repeated administration (60).

In pigs and chickens an increased elimination rate was observed after repeated administration. (60)

The major excretory route seemed to be via the kidneys in all species studied. Hens excreted small amounts of 2,4-D with the eggs. (60)

A gas chromatographic method for the determination of residues of 2,4,5-trichlorophenoxyacetic acid and its propylene glycol butyl ether esters in tissues and fluids is described (59). Both compounds were converted to the methyl ester of 2,4,5-T and analyzed by microcoulometric gas chromatography using a column of 15% Dow 710 on Chromport XXX. Average recoveries of 2,4,5-T added to fat, lean tissue, urine, and blood levels from 0.05 ppm to 20 ppm were 89.3, 89.6, 93.0, and 93.6%, respectively. Corrected recovery of unmetabolized ester added to fat, lean tissue, urine, and blood at levels from 0.5 ppm to 20 ppm averaged 77.9, 70.5, 94.2, and 92.5%, respectively.

Herbicide residues in blacktail deer was studied by Newton and Norris (61). Their report summarizes an exploratory study designed to gain some order-of-magnitude estimates of herbicide residues in various organs of blacktail deer whose habitat was entirely treated either with 2,4,5-T or atrizine. 2,4,5-T was applied at the rate of two pounds per acre acid equivalent as the isooctyl ester, with a small amount of 2,4-D in mixture, in ten gallons fuel oil. Essentially no rain fell during the sampling period.

Several deer were killed in each area at irregular intervals after treatment in hopes of obtaining an estimate of cumulative effects, elimination patterns and reduction of intake with time after treat-

ment. From each deer were taken samples of tissue from brain, thyroid, mesentery lymph nodes, spleen, heart, lung, liver, kidneys, blood, muscle, urine, feces and stomach contents. Mammary glands were sampled on pregnant does. Most of the deer were not fat enough to provide samples of adipose tissue.

It is clear that deer do not accumulate large amounts of either herbicide when exposed to maximum dosages throughout their habitats. Intestinal contents provide abundant evidence of present or past exposure, but low levels of herbicides in most body tissues is evidence of breakdown within the animal, perhaps within some endocrine glands, or passage through the digestive system.

These results are definitely not conclusive. They provide fragmentary evidence that (1) deer do not leave areas thus treated, (2) safe limits for wildlife were apparently observed in these operations, (3) deer do not accumulate 2,4,5-T and atrazine to an appreciable degree, (4) that concentrations in flesh rarely reach detectable levels, particularly in the case of 2,4,5-T, and (5) this ruminant is able to degrade these herbicides almost completely soon after ingestion.

F. Residues in Food

Faust (1964) (69) in a survey of water pollution hazards to man from organic pesticides, came to the conclusion that there did not appear to be danger to health at the present time from the

background concentration of pesticides believed to be in ground and surface water. However, 2,4-D could persist in lake water and bottom mud for long periods under certain environmental conditions. Work in Russia, quoted by Faust, suggested that the threshold taste and odour concentrations auxin compounds especially of phenolic derivatives such as 2,4-D, that would prove unacceptable to the consumer were very considerably below the threshold concentrations for toxic effects. A particular risk might be supposed to lie in contaminated milk drawn from cows feeding in treated pasture, but no residues of either 2,4-DB or 2,4-D were found in the milk of cows that had been fed these compounds (72).

Authorization to use 2,4,5-T on food crops depends on demonstrating that no residue exists in the edible product at harvest (58). The following studies illustrate the amounts of 2,4,5-T that may persist in food crops at various intervals after treatment. When 2,4,5-T was applied to apples as a spray concentration of 40 ppm, residue in the fruit had fallen to 0.004 ppm in 22 days. (68) The application of 2,4,5-T to blueberries at 1 pound per acre resulted in a concentration in the fruit of 0.05 to 0.33 ppm 44 days after application although none was found 733 days after application (71). No detectable 2,4,5-T (sensitivity = 0.01 ppm) was found in rough rice 50 days after applying 2.25 pounds per acre of 2,4,5-T (70). The rice straw contained 0.18 to 1.04 ppm 2,4,5-T 50 days after but not 84 days after application.

Further evidence that very little 2,4,5-T gets into food is seen in results of assays of raw agriculture products and in the Market Basket Survey samples. From about 10,000 food and feed samples examined from 1964 through 1969 only 25 contained trace amounts of 2,4,5-T (less than 0.1 ppm) and only two contained measurable amounts, 0.19 ppm in a sample of milk in 1965 and 0.29 ppm in a sample of sugar beets in 1966 (65). Furthermore, of the 134 total diet samples involving 1600 food composites (Market Basket Survey) analyzed from 1964 through April 1969, only three contained 2,4,5-T. Two were dairy products containing eight to 13% fat with 0.008 and 0.19 ppm in the fat. A single meat, fish and poultry composite from Boston consisting of 17 to 23% of fat was found to contain 0.003 ppm 2,4,5-T on a fat basis (65, 64, 66).

It is concluded from the foregoing that: (1) the herbicide 2,4,5-T does not accumulate in any compartment of the biosphere. (2) The risk of human exposure to 2,4,5-T in food, air or water is negligible (58).

From the very nature of their use, it is unlikely that auzin herbicides will appear as significant residues in food crops. Williams (1964) (73) was unable to detect any residues of auxin herbicides in a number of total diet samples down to the limit of sensitivity (0.01 ppm) of his analytical techniques. Duggan and Weatherwax (1967)

(67) calculated pesticide chemical residues in "total diet" samples collected on 46 days in 25 American cities during a 699 day period from June 1964 to April 1966. Each sample represented the total amount of food and drink consumed by one person over a two-week period. The total samples represented in all a food and drink supply sufficient for 644 days. Herbicide chemicals were found infrequently and averaged about 0.01 mg/day of which one third was 2,4-D and half was MCPA and pentachlorphenol (PCP) combined 2,4-D was found in oils and fats (0.001 mg in 1964/1965) and sugars and sugar products (0.004 mg. in 1964/1965),(0.002 mg. in 1965/1966), while MCPA was found in grain and cereals (0.002 mg. in 1964/1965), in dairy products (0.003 mg in 1965/1966), and in leafy vegetables (0.001 mg. in 1965/1966). These amounts are substantially below the limits set for acceptable daily intake by the World Health Organization and United Nations committees. It seems probable, therefore, that toxic hazards from auxin herbicide residues in food are very small (72).

REFERENCES

A. References: Residues in Soil

1. Akamine, E. K., 1951. Persistence of 2,4-D toxicity in Hawaiian soils. Bot. Gas. 112, pp. 312-319
2. Backelard E. P., 1971. A study of the persistence of herbicides in soil. Weed Abstracts 20(1):59
3. Flieg, O.; Pfaff C., 1951. Movement and decomposition of 2,4-D in the soil, also it's influence on microbiological transformations. Lands Forsch 3, pp. 113-122
4. Freed, V. H., and M. L. Montgomery, 1963. The metabolism of herbicides by plants and soils. In Residue Rev. 3:1-18.
5. Hernandez, T. P. and G. F. Warren, 1950. Some factors affecting the rate of inactivation and leaching of 2,4-D in different soils
6. Jensen, H. L. and H. I. Peterson, 1952. Decomposition of hormone herbicides by bacteria. Acta. Agric. Scand. 2, 215-231.
7. Loos, M. A., 1969. Phenoxyalkanoic acids. In degradation of herbicides, Kearney, P. C., and D. D. Kaufman (Ed.) Marcel Dekker, New York. pp. 1-49.

8. Montgomery, M. L. and L. A. Norris, 1970. A preliminary evaluation of the hazards of 2,4,5-T in the forest environment. USDA Forest Service Research Note PNW 116.

9. Newman, A. S., and J. R. Thomas, 1950. Decomposition of 2,4-dichlorophenoxyacetic acid in soil and liquid media. Soil Sci. Soc. Amer. Proc. 14:160.

10. Norman, A. G., and A. S. Newman, 1950. The persistence of herbicides in soils. Proc. Northeast Weed Control Conf. 4:7.

11. Norris, Logan A., 1966. Degradation of 2,4-D and 2,4,5-T in forest litter. J. Forestry 64(7):475-476.

12. Norris, Logan A., and D. Greiner, 1967. The degradation of 2,4-D in forest litter. Bull. Environ. Cont. & Tox. 2:65-74.

13. Norris, L. A., 1967. Chemical brush control and herbicide residues in the forest environment. In herbicides and vegetation management, pp. 103-123. School of Forestry, Oregon State Univ., Corvallis, Oregon.

14. Norris, Logan A., 1970. Degradation of herbicides in the forest floor. In tree growth and forest soils, pp. 397-411. Oregon State Univ. Press, Corvallis, Oregon.

15. Norris, Logan A., 1971. The behavior of herbicides in the forest. Mimeo, U.S. Forest Serv., Pacific N.W. Forest and Range Experiment Station, Mimeo, 24 pp.

16. Steenson, T. I. and N. Walker, 1956. Observations in bacterial oxidation of chlorophenoxyacetic acids Plant and Soil 8:17.

17. Wiese, A. F., and R. G. Davis, 1964. Herbicide movement in soil with various amounts of water. Weeds 12:101-103 Illus.

B. References: Residues in Water

18. Bailey, G. W., A. D. Thurston, Jr., J. D. Pope, Jr., and D. R. Cochrane, 1970. The degradation kinetics of an ester of silvex and the persistence of silvex in water and sediment. Weeds 18(3):413-418.

19. Brown, E., Nishioka, Y. A., 1967. Pesticides in selected western streams--A contribution to the national program. Pesticides monitoring J. 1(2):38-46.

20. Cochrane, D. R., J. D. Pope, Jr., H. P. Nicholson and G. W. Bailey. The persistence of silvex in water and hydrosoil. Water resources Res. 3:517-523. 1967.

21. Frank, P. A., R. J. Demint, R. D. Comes, 1970. Herbicides in irrigation water following canal-bank treatment for weed control. Weed science 18(6):687-692.

22. Frank, P. A., and R. D. Comes, 1967. Herbicidal residues in pond water and hydrosoil. Weeds 15:210-213.

23. Goerlitz, Donald F., and William L. Lamar, 1967. Determination of phenoxy acid herbicides in water by electron capture and microcoulometric gas chromalography. U.S. Geol. Surv., Water Supply Pap. 1817-C. 21 pp.

24. Krammes, Jay S., and David B. Willets, 1964. Effect of 2,4-D and 2,4,5-T on water quality after a spraying treatment. U.S. Forest Service, Pacific SW Forest and Range Exp. Station Res. Note PSW-52. 4 pp.

25. Lee, G. A., and H. P. Alley, 1970. Exhudation of picloram and 2,4-D from Canada thistle roots. 1970 Res. Prog. Report West. Soc. Weed Sci. pp. 101-102.

26. Montgomery, Marvin L., and Logan A. Norris, 1970. A preliminary evaluation of the hazards of 2,4,5-T in the forest environment. U.S. Forest Serv., Pacific NW Forest and Range Exp. Sta. Res. Note PNW-116. 11 pp.

27. Norris, Logan A., 1967. Chemical brush control and herbicide residues in the forest environment. In herbicides and vegetation management, pp. 103-123. Oregon State University, Corvallis, Oregon.

28. Norris, Logan A., 1968. Stream contamination by herbicides after fall rains on forest lands. Res. Prog. Report, West. Soc. Weed Sci., pp. 33-34.

29. Norris, Logan, A., and Duane G. Moore, 1970. The entry and fate of forest chemicals in streams. Symp. Proc., Forest Land Uses and Stream Environment, pp. 138-158. School of Forestry and Dept. Fisheries and Wildlife, Oregon State University Corvallis, Oregon.

30. Norris, Logan A., 1971. The behavior of herbicides in the forest. U.S. Forest Service, Pacific NW Forest and Range Exp. Sta. 24 pp. mimeo.

31. Norris, Logan A., 1971. Chemical brush control: assessing the hazard. J. Forestry 69(10):715-720.

32. Robson, T. O., 1968. Some studies of the persistence of 2,4-D in natural surface waters. Proc. 9th Brit. Weed Contr. Conf. pp. 404-408.

33. Smith, G. E., and D. G. Ison, 1967. Investigation of effects of large-scale applications on aquatic fauna and water quality. Pestic. Monit. J. 1(3):16-21.

34. Sooper, W. E., I. C. Reignor, and R. R. Johnson, 1966. Effect of phenoxy herbicides on riparian vegetation and water quality. Weeds, trees and turf, January 1966. pp. 8-10.

35. Thomas, Richard E., Jesse M. Cohen, and Thomas W. Bendixen, 1964. Pesticides in soil and water, an annotated bibliography. U.S. Dept. Health, Education and Welfare, PHS Publ. No. 999-WP-17. 90 pp.

36. Wilson, James G. (Chmm.), 1971. Report of the advisory committee to the Administrator of the Environmental Protection Agency. 75 pp., mimeo.

C. References: Residues in Plants.

37. Audus, L. J., 1964. The physiology and biochemistry of herbicides. Acad. Press, New York, 555 pp.

38. Basler, E., 1964. The decarboxylation of phenoxyacetic acids by excised leaves of woody plants. Weed Sci. 12:14-16.

39. Crafts, A. S., 1961. The chemistry and mode of action of herbicides. Interscience Publ. New York 269 pp.

40. Fitzgerald, C. H., C. L. Brown, and E. G. Beck, 1967. Degradation of 2,4,5-trichlorophenoxyacetic acid in woody plants. Plant Physiol. 42:459-460.

41. House, William B., et al., 1967. Assessment of ecological effects of extensive or repeated use of herbicides. Midwest Res. Instit., Kansas City Missouri 369 pp.

42. Kearney, P. C., A. I. Ivensee, C. S. Helling, E. A. Woolson, and J. R. Plimmer, 1972. Environmental significance of the chlorodioxins. Abstracts, Weed Sci. Soc. Amer. p. 14.

43. Klingman, D. L., et al., 1966. Residues in the forage and in the milk from cows grazing on forage treated with esters of 2,4-D. Weeds 14:164-167.

44. Loos, M. A., 1969. Phenoxyalkanoic acids. In degradation of herbicides, pp. 1-49, Kearney, P. C. and D. D. Kaufman (Ed.). Marcel Dekker, Inc. New York.

45. Morton, H. L., 1966. Influence of temperature and humidity on foliar absorption, translocation, and metabolism of 2,4,5-T by mesquite seedlings. Weeds 14:136-140.

46. Morton, Howard L., E. D. Robison, and Robert E. Moyer, 1967. Persistence of 2,4-D, 2,4,5-T and dicamba in range forage grasses. Weeds 15:268-271.

47. Montgomery, Marvin L., and Logan A. Norris, 1970. A preliminary evaluation of the hazards of 2,4,5-T in the forest environment. U.S. Forest Serv., Pacific NW Forest and Range Exp. Sta. Res. Note PNW-116. 10 pp.

48. Norris, L. A., 1967. Chemical brush control of herbicide residues in the forest environment. In herbicides and vegetation management, pp. 103-123.

49. Norris, Logan A., 1971. Chemical brush control: assessing the hazard. J. Forestry 69(10):715-120.

50. Shaw, W C., J. L. Hilton, D. E. Moreland, and L. L. Jansen, 1960. The fate of herbicides in plants. In the nature and fate of chemicals applied to soils, plants, and animals, pp. 119-133, Symp. Proc., USDA, Agric. Res. Serv., Beltsville, Md.

51. Sopper, W. E. et al., 1966. Effect on phenoxy herbicides on riparian vegetation and water quality. Weeds, Trees and Turf.

52. Wilson, James G. (Chmn), 1971. Report of the Advisory Committee on 2,4,5-T to the Administrator of the Environmental Protection Agency. 75 pp., mimeo.

D. References: Residues in Air

53. Courtney, K. D. and J. A. Moore, 1971. Teratology studies with 2,4,5-T and tetrachlorodioxin. Submitted to Toxic. Appl. Pharmacol.

54. Khera, K. S., B. L. Huston and W. P. McKinley, 1971. Pre- and posnatal studies on 2,4,5-T, 2,4-D and derivatives in Wistar rats. Toxic. Appl. Pharmacol., in press.

55. Moore, J. A. and K. D. Courtney, 1971. Teratology studies with the trichlorophenoxyacid herbicides 2,4,5-T and Silvex. Teratology in press.

56. Norris, L. A., 1967. Chemical brush control and herbicide residues in the forest environment. In herbicides and vegetation management in forest ranges and noncrop lands, pp. 103-123. Oreg. State Univ., Corvallis.

57. Norris, L. A., and J. Zavitkovski. Unpublished data, School of Forestry, Oregon State University.

58. Report of the Advisory Committee on 2,4,5-T to the Administrator of Environmental Protection Agency.

E. References: Residues in Animals

59. Clark, D. E., 1969. Butyl ether esters in animal tissue blood, and urine. J. Agr. Food Chem. 17(6):1168-1170.

60. Erne, K., 1966. Distribution and elimination of chlorinated phenoxyacetic acid in animals. Acta. Vet. Scand:7:240.

61. Newton, Michael and L. A. Norris, 1968. Herbicide residues in blacktail deer. From Forests treated with 2,4,5-T and atrizine.

62. St. John, L. E., Wagner, D. G., and Lisk, D. J. 1964. Fate of atrizine, kuron, silvex, and 2,4,5-T in the dairy cow. J. Dairy Sci. 47:1267-1270.

63. Zielinski, Walter L., and Fishbein, Lawrence, 1967. Gas chromatrographic measurement of disappearance rates of 2,4-D and 2,4,5-T acids and 2,4-D esters in mice J. Agr. Food Chem. 15:841-844, illus.

F. References: Residues in Food

64. Corneliussen, P. E., 1969. Pesticide residues in total diet samples. Pesticide Monit. J., 2:140-152.

65. Duggan, R. E., 1971. Memorandum to Wayland J. Hayes, Unpublished, March 12, 1971.

66. Duggan, R. E., H. C. Barry, and L. Y. Johnson, 1967. Pesticide residues in total diet samples. Pesticide Monit. J., 1:2-12.

67. Duggan, R. E. and J. R. Weatherwax. Dietary intake of pesticide chemicals. Science 157, 1006 (1967)

68. Edgerton, L. J. and D. J. Lesk, 1963. Determination of residues of 2,4,5-trichlorphenoxyacetic acid in apples by radioiostopes and gas chromatographic methods. Proc. Am. Soc. Hort. Sci., 83:120-125.

69. Faust, S. D. Pollution of the water environment by organic pesticides. Clin. Pharmacol. Therap. 5, 677 (1964).

70. Syracuse University Research Corporation, 1970. 2,4,5-T residues in rough rice and straw. Unpublished data. Cited in Dow communication dated Jan. 19, 1971.

71. Trevett, M. F., 1964. A request for approval of a contact method of applying 2,4-D and 2,4,5-T for control of woody weeds in Maine lowbush blueberry fields. Unpublished data. Cited in Dow communication dated Jan. 19, 1971.

72. Way, J. M, 1969. Toxicity and hazards to man, domestic animals and wildlife. From Some Connomly Used Auxin Herbicides. Residue Reviews, Vol. 26.

73. Williams, S. Pesticide residues in total diet samples. J. Assoc. Office. Agri. Chemists 47, 815 (1964)

Section VI

ENVIRONMENTAL IMPACTS OF THE PHENOXY COMPOUNDS 2,4-D, 2,4,5-T, and 2,4,5-TP

A. Hazards to Man

There is no evidence of harmful effects on man being caused by any of the three phenoxy compounds when used properly and in the manner prescribed on forest and range vegetation. Human exposure to an environmental chemical such as 2,4,5-T depends on (1) pattern of usage, i.e., how widely and frequently applied and in what amounts and, (2) its fate in the environment, i.e., does it accumulate or is it degraded as fast as applied. These herbicides offer minimal hazard to man and his environment under forest and range use, because large and prolonged doses required to cause significant biological effects do not occur.

The principal routes of toxicity to man are either orally or by inhalation; there appears to be little hazard of transport through the skin although individual allergies can develop leading to dermatitis (Vallet 1965)(A6). Eyes may be directly but are usually only temporarily affected. Hazards to man may occur from the concentrated chemical before dilution, from inhalation of spray or dust during application, or from ingestion of the chemicals in food or in water. Because the greatest hazards are from the concentrated chemical and because man is handling the chemicals

in this form at all stages from manufacture to dilution, it follows that he is at greater potential risk than any other organism. However, there are very few reports in the literature of tests or incidents of poisoning of man by these compounds, the majority of these reports refer to accidental poisoning of children. As a result it is now generally accepted that auxin-type 1/ herbicides do not present a direct toxicity hazard to man (Barnes 1965)(A1) when correctly handled or used for weed control.

Kraus in 1945 (in Kephart, 1945)(A7) reported that he had taken 0.5 g. of 2,4-D per diem for 21 days with no demonstrable ill effects. A clinical study was made in Denmark by Nielsen et al. (1965)(A5) on a 23-year old man who had committed suicide by apparently drinking 125 ml. of 50 percent w/v 2,4-D dimethylamine salt. The total weight of 2,4-D in his body was calculated as being not less than six g. (the equivalent of 80 mg./kg.), about 10 percent of the total weight of active material ingested. The principal damage appeared to be to nerve tissues and the central nervous system (A).

Edwards and Ripper (1953)(A2) have discussed the hazard to operators from inhalation of sprays and aerosols during application of herbicides with particular reference to methods of protection. Monarca

1/ Auxin. Any group of substances which promote plant growth by cell elongation, bring about root formation or cause bud inhibition or other effects. 2,4-D, 2,4,5-T and 2,4,5-TP are compounds of this group.

and Dr. Vito (1961)(A4) have described a clinical study of an acute case of accidental poisoning of a man in Italy. In this instance a farmer became ill after applying a 40 percent aqueous solution of 2,4-D by handpump against the wind. He was admitted to hospital, suffered a relapse after 18 days, and recovered sufficiently to be discharged after 40 days. Initial symptoms of muscular weakness, vomiting, perspiring freely, and oliguria were noted in the field while a diagnosis of bradycardia, respiratory difficulties, and urinary abnormalities was made after admission to hospital. However, the authors report that the case was exceptional (A). Fetisov (1966)(A3) has reported similar field symptoms in Russian workers engaged in field applications of 2,4-D. This author concluded that a range of formulations of 2,4-D was "highly toxic to animals in different ways of introduction." While reports of minor discomfort following exposure to auxin sprays during field application are rarely reported in scientific literature, there is no doubt that a proportion of workers so exposed do suffer a degree of transitory discomfort. Whether this is of any significance as long-term toxic hazard has not been determined for man (A).

There has been some alarm (perhaps unjustified) about the human toxicity resulting from the use of 2,4-D and its derivatives. Some of the case histories of persons contracting neuropathy as a result of 2,4-D treatment are presented here to permit the reader

to form his own opinion about the magnitude of the hazard associated with the use of 2,4-D compounds. (B) Goldstein et. al. (1959)(B) in their report on peripheral neuropathy after skin exposure to an ester of 2,4-D state that three individual patients, two farmers and a female bookkeeper, suffered the disorder some hours after exposure to the 2,4-D formulation while attempting to kill weeds. The symptoms progressed through a period of days until pain, paresthesias and parlysis were severe. Diability was protracted and recovery was incomplete even after a lapse of years. They concluded that there was little doubt that the symptoms resulted from the percutaneous absorption of the 2,4-D. The electromyographic examinations supported the diagnosis of peripheral neuropathy. Berkley and Magee (1963)(B) report a similar case of neuropathy in a 39 year old farmer four days after exposure to 2,4-D dimethylamine salt; the symptoms included numbness and incoordination of the hand and finger muscles and a slow recovery. These authors conclude that persons who get peripheral neuropathy from exposure to 2,4-D are very rare compared to the number of exposures there must be. They state that some individuals may have a predisposition to neuropathy and suggest that all users of these herbicides use protective clothing and wash immediately with soap and water in case of accidental exposure.

Mitchell (1946) quotes the experimental work of E. J. Kraus concerning the ingestion of 500 mg of purified 2,4-D/day by a man over a period of 21 days without ill effect. Seabury (1963) reports on the administration of the sodium salt of 2,4-D to two patients suffering from

coccidioidomycosis. The first patient received an intramuscular injection of 2.0 g without any toxic reactions. The second patient received 3.6 g by parenteral injection; there were severe toxic reactions including coma, fibrillary twitching of some muscles, hyporeflexia and urinary incontinence. Recovery from the toxic effects of the injections were complete in 48 hours but the patient died of his disease 17 days after the injection (B).

According to DiPalma (1965)(B), a man committed suicide by consuming about 6.5 g of 2,4-D; from this and the other information, it appears that the lethal dose for a human lies in the range of 50 to 100 mg/kg (B).

The Dow Chemical Co. (C1) has prepared an extensive health inventory of 126 manufacturing personnel in an effort to identify adverse effects of inhaled 2,4,5-T. The inhalation rate of the agent was estimated to be 1.6 to 8.1 mg/day per worker, depending on the work assignment, for periods of up to three years and at total career exposures in excess of 10,000 mg. The survey indicates that no illness was associated with 2,4,5-T intake. Specifically there was no increase in skin ailments or of alkaline phosphatase or SGPT levels as compared with controls having no exposure to 2,4,5-T.

The result was entirely different in a plant where the 2,4,5-T produced contained a high proportion of dioxin (TCDD). The latter plant was studied by Bleibert in 1964 (C2) and again six years

later by Poland et. al. (C3) who also reviewed earlier studies in factories in other countries where TCDD had been a problem. Poland and associates reported on 73 employees whose health was found to be improved compared to that of workers in the plant six years earlier. Eighteen percent of the men had suffered moderate to severe chloracne, the intensity of which correlated significantly with the presence of residual hyperpigmentation, hirsutism, and eye irritation and with a high score on a test indicating a manic reaction. The chloracne did not correlate with job location or duration of employment at the plant or with coproporphrin excretion. One of the men had uroporphyrinuria but, unlike the situation six years earlier, no porphyria could be found. Systemic illness such as may be produced by TCDD was markedly less than that reported in previous studies of 2,4,5-T plants and probably no greater than expected in unexposed men of the same age. (C)

As far as occupational exposure is concerned it is clear that any danger of 2,4,5-T formulations residues in their TCDD content. The primary manifestation of industrial TCDD intoxication is chloracne, an easily detected, in fact highly disfiguring, dermatitis. It is significant that this condition has not been a problem in factories producing 2,4,5-T with a low content of TCDD, nor among persons who apply the herbicide as a part of their regular occupation. It is therefore highly unlikely that exposure to traces of TCDD will have any effect on persons who use 2,4,5-T formulations occasionally or who merely encounter possible traces of it in the environment.(C)

Data are too limited for a firm conclusion but there is no evidence to suggest that TCDD as a contaminant in 2,4,5-T is likely to be encountered by animal or man in sufficient dosage to cause toxic reactions.

No proven instance of toxicity associated with 2,4,5-T intake in man has been found in industrial or agricultural workers known to have had repeated, relatively high levels of exposure to 2,4,5-T of low dioxin content; and the safety factor for the general population is estimated to be several orders of magnitude greater than that of 2,4,5-T factory workers (C).

The very small number of cases in which human ingestion of 2,4,5-T led to clinical illness offer no information on the minimal dosage of the compound that is toxic to man. In animals, however, the toxicity of 2,4,5-T is similar to that of 2,4-D, consequently some information on 2,4-D is of interest. When 2,4-D was investigated as a possible treatment for disseminated coccidiodomycosis, the patient had no side-effects from 18 intravenous doses during 33 days; each of the last 12 doses in this series was 800 mg (about 15 mg/kg) or more, the last being 2000 mg (about 37 mg/kg). A 19th and final dose of 3600 mg (67 mg/kg) produced mild symptoms.(C4) Suicidal ingestion of a quantity of 2,4-D as a single dose known to be greater than 6500 mg (in excess of 90 mg/kg) was fatal (C5).

The acid of silvex is appreciably irritating to the eyes and skin, paricularly in high concentrations. The undiluted esters may cause painful but temporary injury to the eyes. Skin irritation may occur from repeated or extended contact with the skin but there is no evidence of toxicity resulting from skin absorption. There was no sign of allergic response to the application of a 1% solution of a commercial formulation of silvex to the skin of 50 human test subjects (B).

B. Hazards to Animals (Domestic and Laboratory)

The toxicity of agricultural chemicals to land fauna is normally quoted in terms of the dose that kills 50 percent of a population of test animals (LD50). While this figure gives a useful indication of the comparative toxicities of different compounds to a given test species, the figures obtained in different tests may be influenced by a number of factors. Thus the age and sex of the test animal, method of dosing, and general conditions of the test may have an important bearing on the susceptibility of the animals to the compound being studied (A). The formulation of the active compound has a considerable influence: for instance, 2,4-D acid has an LD_{50} to rats of 375 mg./kg. but the sodium salt has an LD_{50} of 805 mg./kg., the propylene glycol butyl ether ester of 570 mg./kg. and the isopropyl ester of 700 mg./kg. (Rowe and Hymas 1954)(A20). It should be noted, however, that Bjorklund and Erne (1966) do not regard these differences as being appreciable. In addition, the LD_{50} for

different test species may vary quite widely: for example, 2,4-D acid has an LD_{50} of 375 mg./kg. for rats, of 100 mg./kg. for dogs, 469 mg./kg. for guinea pigs, and 541 mg./kg. for chicks (Rowe and Hymas 1954)(A20).

Erne (1966)(A13) studied the distribution and elimination of 2,4-D and 2,4,5-T in these animals. Amine and alkali salts of both compounds were readily absorbed and completely distributed in the body, but 2,4-D ester was incompletely absorbed and reached only a low level in the plasma and tissues. The highest tissue levels of the two compounds were found in liver, kidney, spleen, and lungs and the levels found in these organs sometimes exceeded the level found in the plasma. In blood cells some 10 to 20 percent of the plasma level was found. Penetration of 2,4-D into placental tissue of pigs was recorded but there was little or no evidence of penetration into adipose tissue or the central nervous system. Elimination of the compounds was rapid, the plasma half-life being about three hours in rats, eight in calves and chickens and 12 in pigs. The tissue half-life values ranged between five and 30 hours. No retention of the compounds was noted in the tissues. There was no accumulation after repeated dosing and in pigs there was an increase in the rate of elimination after repeated administration. In all species, the main excretory route was via the kidneys. Khanna and Fang (1966)(A16) traced the metabolism of C^{14} labeled 2,4-D in rats dosed at rates from one to 100 mg./animal. Radioactivity was found in all the organs studied together with some accumulation as early as one hour after

dosing. At the one mg. dose rate a concentration peak of radioactivity was demonstrated after six to eight hours but decreased thereafter and was non-detectable by 24 hours. At the 80 mg. dose the peak occurred at eight hours and persisted for 17 hours. Extracts of the tissues were shown to contain mainly unchanged 2,4-D residues. No radioactivity was found in the expired carbon dioxide, but elimination in urine and faeces was dose dependent. At the one to 10 mg. doses 93 to 96 percent of the ingested 2,4-D was excreted unchanged in the urine in the first 24 hours. At the 20 to 100 mg. doses greater amounts of 2,4-D were found in the second 24 hour period after dosing, with a linear decrease in percentage recovery with increase in dose. In experiments with cattle Gutenmann et. al (1963)(A15) were unable to detect any residues of 2,4-DB or 2,4-D in milk or faeces of cows fed five p.p.m. of either compound in a 50 pound daily ration. In these experiments there was no evidence of beta-oxidation of 2,4-DB to 2,4-D. Disappearance of 2,4-D was thought to occur as a result of dilution in the rumen, some absorption on the gut wall, and by decomposition. In subsequent experiments Bache et. al. (1964)(A23) and St. John et. al. (1964)(D) studied the fate of MCPA, MCPB, 2,4,5-T, and a number of other herbicides in cattle. All the MCPA fed to a single steer (113.5 mg. single dose based on five p.p.m. of a 50 pound daily ration) was accounted for in its urine over the four days after administration. These authors discussed the significance of biological active residues of auxin compounds in animal excreta that might become incorporated in manure or straw. It was shown that 2,3,6-TBA residues in particular could remain active for a period of months and affect susceptible

crops to which the contaminated manure was applied. The same hazard does not normally exist with the phenoxyacetic acid derivatives, where the compounds are broken down and become biologically inactive in a relativaly short period of time. However, Lisk (1966)(A17) has pointed out that the excretion of 2,4-D in the urine of cows does present the admittedly remote possibility of active 2,4-D being transferred from a treated pasture to a susceptible crop.

Mitchell (in Kephart 1945)(A7), Dalgaard-Mikkelsen et. al. (1959)(A10) and Goldstein and Long (1960)(A14) all reported that there were no apparent ill-effects in cattle, sheep, or horses from grazing pasture sprayed at herbicidal or two times herbicidal rates of 2,4-D or MCPA. Grigsby and Farwell (1950)(In Springer 1957)(A22) reported that there was no significant difference in the amount of feeding of horses, cows, sheep, and pigs in untreated plots or plots sprayed with the sodium salt or the isopropyl ester of 2,4-D or the isopropyl ester of 2,4,5-T. However, there did appear to be less feeding in plots sprayed with the alkanolamine salt of 2,4-D. There was no effect on milk production of cows feeding on sprayed vegetation. Goldstein and Long (1960)(A14) found no ill-effects on two cattle from adding 0.25 pints of a 1.5 percent w/v 2,4-D/2,4,5-T mixture to every five gallons of their drinking water for 41 days. These authors also reported spraying the skins of a calf, of a cow, of sheep, and of pigs with doses ranging from 0.002 to 0.008 pounds of 2,4-D or 2,4-D/ 2,4,5-T mixture, with no ill-effects. These dose rates would be of the order of those that might occur in an instance of spray drift.

Dobson (1954)(A11) sprayed 2,4-D, or 2,4,5-T on grassed chicken runs daily for 14 days at normal and ten times normal dose rates. 2,4,5-T significantly reduced egg production and the weight of the birds; 2,4-D affected egg production, mainly in the second week of spraying or during the week after spraying had stopped. In all instances there was no effect on the fertility of the eggs and all the progeny reared well, although the dose rates and frequency of application in this trial were much more severe than are likely to be found in practice. Erne (1966)(A13) showed that some of the 2,4-D fed to hens could be excreted in their eggs. Dunachie and Fletcher (1967)(A12) injected hen's eggs with 2,4-D, MCPB, 2,4-DB, and 2,3,6-TBA amongst a range of other herbicides. Dose rates were 10, 100, and 200 p.p.m. equivalent to 0.5, 5, and 10 mg./egg. The percentage hatch was recorded. At the lowest dose there was 90 percent from the TBA-treated eggs, and 80 percent from the 2,4-D treated eggs. At the highest dose there was 50 percent hatch from 2,4-D and TBA. None of the chicks that hatched were deformed although some feather blanching was noted from the 2,4-DB treatments. Roberts and Rogers (1957)(A19) reported on various feeding experiments on turkeys with alfalfa sprayed with a low volatile ester of 2,4,5-T at herbicidal rates. No deleterious effects were noted. Calculations were quoted to show that for a one kg. chicken to acquire a lethal dose of 2,4-D from an application rate of one pound/acre, the bird would have to consume in two days all the 2,4-D applied to the vegetation over an area of 72 square feet.

Accounts are given of direct oral dosing or dermal applications of auxin herbicides to a variety of domestic animals by Kephart (1945)(A7) (cow), Rowe and Hymas (1954)(A20) (laboratory animals and cattle), Dalgaard-Mikkelsen et. al. (1959)(A10) (heifers), Palmer (1963)(A18) (cattle), Clarke et. al. (1964)(A9) (sheep), and Strach and Bohosiewicz. (1964)(A21)(pigs). Palmer (1963)(A18) gave daily oral doses of 2,4-D alkanolamine salt to steers for five days in every seven. He recorded signs of poisoning in animals dosed at 250 mg./kg. after 15 administrations as opposed to 86 administrations of 100 mg./kg.; at 50 mg./kg. no ill-effects were recorded over a period of 112 administrations. From these results he concluded that although animals could probably ingest enough 2,4-D from concentrated solutions at any one time to produce illness or death, the chronic toxicity of the compound was sufficiently low to make it unlikely that an animal would pick enough of it over a period time to cause any serious ill-effects. Further work by Palmer and Radeleff (1964)(B) using single animals gave the following results:

1. Sheep tolerated 481 daily doses of 100 mg./kg. of the alkanolamine or propylene glycol butyl ether ester of 2,4-D.

2. Cattle suffered from chronic typanites after 88 daily doses of 100 mg./kg. of the alkanolamine salt of 2,4-D. One animal died after 34 daily doses of 200 mg./kg.

3. Sheep tolerated 481 daily doses of 100 mg./kg. of the triethylamine salt of 2,4,5-T but succumbed to 369 doses of 100 mg./kg. of the propylene glycol butyl ether ester.

4. Sheep were killed by 383 daily doses of 100 mg./kg. of MCPA amine.

Strach and Bohosiewicz (1964)(A21) reported that no abnormal behavior in pigs had been noted following 40 daily doses of 15 to 100 mg./kg. of 2,4-D, nor from single doses of 2,4-D of 200 to 800 mg./kg.

In short term trials by Bjorklund and Erne (1966)(A8), calves and pigs showed definite though reversible symptoms of poisoning after single doses of 2,4-D of 200 and 100 mg./kg. respectively. Rats and fowls did not show any sign of distress after single doses of 100 and 300 mg./kg., respectively, and fowls tolerated daily doses of 300 mg./kg. daily in their feed for several weeks without visible effects. Repeated daily doses of 50 mg./kg., however, led to toxic symptoms in some pigs. In longer term studies (Erne 1966)(A13), five young pigs were fed 500 p.p.m. of 2,4-D for up to 12 months but, although various toxic effects were noted and their growth rate was affected, none of the animals died. When 2,4-D was fed to a sow throughout gestation and for a further six weeks, 10 of the 15 underdeveloped and apathetic piglets she produced died within 24 hours and the mother subsequently had to be slaughtered because of abnormalities that developed in her spine. Heavy dosing of pregnant rats, however, with 1000 p.p.m. of 2,4-D in their drinking

water over 10 months and of their off-spring for up to two years, while leading to retarded growth and increased mortality, did not produce unequivocal signs of toxicity. Continued administration of 500 p.p.m. of 2,4-D in feed or 1,000 p.p.m. in the drinking water of fowls led to reduced egg production and kidney abnormalities. These results led the authors to conclude that the chronic toxicity of 2,4-D to the species studied was moderate. They were, however, concerned about the mortality of new-born piglets, with evidence of movement of 2,4-D through the placental tissues, and the reduced egg production in fowls which they thought might indicate a possible interference with reproductive processes.

In general the findings of other workers support these conclusions on acute and chronic toxicity. In all the work quoted the amounts administered to the test animals for effect, have been well in excess of the amounts they might be expected to pick up from a treated pasture, or in feed derived from crops that had at some time been treated with auxin herbicides at normal dose rates. (A).

Dr. O. G. Fitzhugh (1967), Toxicological Advisor, Division of Toxicological Evaluation, Food and Drug Administration, writes that the FDA laboratories have conducted a three generation, six litter reproduction test in rats: (B)

1. In the two-year feeding test on dogs there were three male and three female dogs in each group. The levels of 2,4-D in the diets were 0, 10, 50, and 500 ppm 2,4-D. There were no

gross or microscopic findings related to 2,4-D. There was no dose-related clinical or hematologic effect. The "no effect" level was greater than 500 ppm (i.e., not determined).

2. In the two-year rat feeding study there were 25 male and 25 female rats per group. The diets of the various groups contained 0, 5, 25, 125, 625, or 1,250 ppm of 2,4-D. There was no effect on growth, survival, organ weights, hematologic values or occurrence of tumors. Neither gross nor microscopic changes were noted. No "no effect" level was found (i.e., greater than 1,250 ppm).

3. In the rat reproduction studies 20 males and 20 females were used (i.e., where there were enough survivors) and they were fed 0, 100, 500, or 1,500 ppm of 2,4-D in their diets. No effect was observed at the 100 or 500 ppm levels. At the 1,500 ppm level, there was no effect on fertility nor on the average number of pups/litter. There were, however, significant effects on the average number (%) weaned and also on the weights of the weanlings (i.e., average weight of survivors). No histopathology was done and the "no effect" level is at least 500 ppm but less than 1,500 ppm of 2,4-D in the diet.

Palmer (1963)(B1) conducted a chronic toxicity test with yearling steers using an alkanolamine salt of 2,4-D (2,4-D Dow Weed Killer

Formula 40). He found that 112 daily doses of 50 mg./kg. of this 2,4-D salt had no deleterious effect on the steer and concluded that it was not accumulated in the steer since doses of 100 and 250 mg./kg. had produced toxic symptoms. Clark (1964)(B2) confirmed this observation by a study on the fate of 2,4-D in sheep using C^{14} labeled 2,4-D. He showed that 96% of the 2,4-D was excreted unchanged in the urine in 72 hours. About 1.4% of the radioactivitiy was found in the feces during this same period. A similar study was reported by Khanna and Fang (1966)(B3) in which they fed C^{14} labeled 2,4-D to rats; they found that the time required for elimination was dependent upon the dose. For example, a 1 to 20 mg./rat dose was 88.8 to 95.6% eliminated in 24 hours. At a dose of 100 mg./rat, 144 hours was required for 75.5% recovery of the radioactivity.

Grigsby and Farwell (1950)(B) sprayed alfalfa and brome grass with two to four times the usual quantities of 2,4-D (sodium salt, alkanolamine salt and isopropyl esters used in separate experiments) and then fed it to sheep, chickens, swine, dairy cows and steers. They concluded that these 2,4-D compounds were not injurious to livestock under these conditions. They did, however, note an off-flavor in the milk. Buck et. al. (1961)(B4) fed herbicide-treated plants in an effort to determine whether the spraying of toxic weeds would make them more palatable to cattle and it did not. There is, however, an authenticated case in which sugar beet leaves accidentally

sprayed with 2,4-D accumulated enough nitrate to become toxic (Stahler and Whitehead, 1950)(B5). This does not seem to be a severe practical problem since sugar beets are very sensitive to 2,4-D, and are not normally sprayed. Some early reports on the increase in HCN content in wild cherry after spraying have not been disproved; instead the level of HCN decreases steadily for 15 days (Lynn and Barrons, 1952)(B6) after application of the 2,4-D.

Atrazine (2-chloro-isopropylamino-6-ethylamino-s-triazine), kuron (propylene glycol butyl ether ester of 2-(2,4,5-trichlorophenoxy) propionic acid), silvex (2-(2,4,5-trichlorophenoxy) propionic acid), and 2,4,5-T (2,4,5-trichlorophenoxyacetic acid) are often used for weed and brush control in the vicinity of forage crops. The reality of contamination of forage by drift or uptake prompted the study of the fate of these herbicides in the dairy cow (D).

No residues of these herbicides were found in the milk. About 2% of intact atrazine was eliminated in the urine. About 67% of the kuron was hydrolyzed and eliminated as silvex (sodium salt) in the urine. Within experimental error, silvex and 2,4,5-T appeared to be totally eliminated in the urine as salts (D).

C. Hazards on Vegetation - Indirect Effects

Indirect effects of herbicides on grazing animals have been associated with increased toxicity of toxic plants, increased palatability of

normally non-palatable toxic plants (e.g., ragwort, Senecio jacobaea) and induced toxicity in normally nontoxic plants (e.g., temporary increases in nitrate content) (Willard 1950)(A40). However, Fertig (1953)(A29) claimed that, up to 1953 in America, in all cases where poisoning of livestock from herbicides had been reported, the effects noted could be attributed to some other cause.

Examples have been given by Willard (1950)(A40) of cattle eating wild cherry (Prunus serotina), of pigs eating Cocklebur (Xanthium sp.), and of lambs eating thistles after herbicidal treatment with auxins. Instances have been reported of ragwort becoming "sweeter" for two or three days after application and being preferentially grazed by cattle for a short period. Grigsby and Ball (1952)(A31) and Lynn and Barrons (1952)(A3) investigated the hydrocyanic acid (HCN) content of the leaves of wild cherry from untreated trees and trees treated with 2,4,5-T. Their conclusions were that the foliage was no more toxic to cattle after treatment and that there might even be less HCN in the leaves of the treated trees than in those of the untreated ones. Buck et. al. (1961)(A26) fed the alkaloid-containing plants Delphinium barbeyi (tall larkspur) and Helenium hoopseii (sneezeweed), after treatment with 2,4-D ester or 2,4,5-T ester, to calves and ewes. No increased toxicity of the plants attributable to application of the herbicides was noted. Williams and Cronin (1963)(A41) analyzed D. Berbeyi, treated

with 2,4,5-T amine at various growth stages, and showed that the alkaloid content of the plants was increased for several weeks after treatment at the vegetative and early bud stages. It was noted, however, that the bitter taste of the alkaloids might make the treated plants even less palatable to animals than untreated ones.

Swanson and Shaw (1954)(A38) showed that 2,4-D affected the HCN content of Sudan grass (Sorghum vulgare ssp. sudanense). Initially there was a decrease in the content of HCN for four days after treatment there was an increase over the controls which was maintained for a further 12 days. Similar effects were shown to occur with the nitrate content of leaves. Buck et. al. (A26) thought that there might be a relationship between HCN and nitrate metabolism in Sudan grass, an increase in one leading to a decrease in the other.

The clinical aspects of nitrate poisoning in stock, conditions under which nitrates are likely to accumulate in the leaves of certain plants, and lists of these plants have been reported by Bradley et. al (1940)(A42), Davidson et. al. (1941)(A28), Gilbert et. al. (1946)(A32), Case (1957)(A27), and Sund et. al. (1960)(A37). The toxic effects of nitrate are caused by a reduction of nitrate to nitrate and the conversion by nitrite of haemoglobin in the blood to methoglobin: the animal dies from asphyxia. Intravenous injection of methylene blue in doses of two g./500 pounds of body weight gives immediate relief.

Nitrate in plants is generally present in the form of potassium nitrate and increases in nitrate content have been associated with drought conditions and high soil nitrogen (Gilbert et. al. 1946, (A32), Case 1957(A27)). Sund et. al. (1960(A37) noted a high nitrate content in Urtica spp. and Rubus spp. after heavy rains, followed by preferential grazing of these and other weed species by cattle. A number of abortions in these cattle was correlated with occurrence of high nitrate rather than grazing of the weed species per se. Recent increases in vitamin A deficiency in North American ruminants has been associated with ingestion of nitrates occurring in herbicide treated plants by Phillips (1964)(A34).

The accumulation of nitrates in the leaves of treated sugar beets is well known (e.g., Savage 1949)(A35). Increased levels of nitrate in the leaves of this crop as a result of herbicide application have been reported by Willard (1950)(A40), Stahler and Whitehead (1950)(A36) and Whitehead et. al. (1956)(A39). Isolated incidents have been reported of nitrate poisoning of cattle in America as a result of feeding on sugar beet that had previously been sprayed. In one incident in N. Dakota, the nitrate content of sugar beet leaves after spraying was found to vary from 1.81 to 8.77 percent of the dry weight, as against 0.22 percent for untreated plants and a toxic level of 1.5 percent (Stahler and Whitehead 1950)(A36).

Cell-free extracts of maize and cucumber from plants that had been previously sprayed with 10 and 100 p.p.m. of 2,4-D were investigated by Beevers and Hageman (1962)(A24). The level of nitrate reductase was increased in maize but reduced in cucumber. Studies on the formation and breakdown of nitrates in plants (Fertig 1952)(A29) Whitehead et. al. 1956 (A39) have shown that 2,4-D causes more rapid increases in nitrate content than MCPA, that levels rise to a peak soon after spraying and subsequently decrease with time, and that increases in light intensity hasten decreases in nitrate content.

Studies on forage crops (Berg and McElroy 1953)(A25) and on a range of weed species (Frank and Grigsby 1957)(A30) have shown which of these may contain high levels of nitrates after auxin application. They also list a large number of plants in which the levels of nitrate do not increase after auxin application.

It is clear from these reports that nitrate poisoning in stock does occur from time to time and that it is possible for the hazard to be increased by application of auxin herbicides to nitrate-accumulating plants.

Livestock managers should make provisions to exclude cattle from sprayed areas for short periods following treatment when the probability of nitrate poisoning exist.

D. Hazards on Insects

Herbicides affect bees (Apis mellifera) and other insects if they kill the plants on which the insects feed (A). In addition, Wahlin (1950)(A53) has reported that 2,4-D and MCPA were toxic to bees, not only from visiting the flowers but also as a result of drinking contaminated water trapped on treated plants. Other workers have reported effects on bees after application of auxin herbicides to plants in flower but not at other times (Haragsimova 1962, A54, Palmer-Jones 1964, A51). Palmer-Jones (1964)(A51) and Antoine (1966)(A44) have suggested that 2,4-D might have some effect on nectar which made it toxic to bees. King (1960a)(A49) has shown that radioactive 2,4-D can be translocated to the nectar of Poinsetta and red clover plants and may be detectable there for two to three days after treatment. Feeding trials of auxin herbicides to been have been reported by Glynne Jones and Connell (1954)(A46), Palmer-Jones (1960)(A51), King (1960b)(A49), and Byrdy (1962)(A45). Palmer-Jones (1964)(A51) found no effect on bees that had been directly dusted with 2,4-D or when they were made to crawl through 2,4-D dust in order to enter the hive. Glynne Jones and Connell (1954)(A46) classed 2,4-D and MCPA as stomach/contact poisons of low toxicity to bees, with LD50 values of 0.015 mg. compared to insecticides in the range 0.00004 to 0.002 mg. Byrdy (1962)(A45), on the other hand, reported total mortality of bees within four days of feeding 30 ug. of 2,4-D and 10 percent mortality within three days rising to 20 percent in five days of

feeding 20 ug. Johansen (1959)(A48) reported that 2,4-D and related compounds were not toxic to bees, except when formulated as the alkanolamine salt or the isopropyl ester.

Occasional observations on other insects have been reported. Maxwell and Harwood (1960)(A50) treated broad bean (Vicia faba) plants with sublethaldoses of 2,4-D and recorded a marked increase in the reproduction of the pea aphid (Macrosiphum pisi) feeding on them. The longevity of adult aphids was unaffected. Robinson (1959)(A52) also recorded increased fecundity in another pea aphid (Acyrthosiphon pisum) after caging on broad bean plants treated with 2,4-D. Adams (1960)(A43) and Adams and Drew (1965)(A43) showed that the application of 2,4-D amine could enhance aphid infestation in New Brunswick grain fields, probably as a result of depressing the activities of coccinellid beetles predating on the aphids. In laboratory experiments with coccinellid larvae treated with 2,4-D amine, there was a fourfold increase in mortality and an increase in time to pupation. There was little mortality amongst the adult beetles, which usually recovered after a few hours inactivity. Ishii and Hiran (1963)(A47) concluded that increases in the growth rate of the larvae of the rice stem berer (Chilo suppressalis) feeding on 2,4-D treated rice plants, was a consequence of increased nitrogen content of the plants rather than a direct effect of the chemical itself.

It appears that there may be some effects to bees from application of auxin herbicides to plants in flower. These effects may be negated by timing of application, size of treatment units and method of application. Otherwise there would seem to be little hazard to insects from direct toxicity of the compounds at normal herbicidal rates of application.

E. Hazard to Soil Fauna

Bollen (1961)(A56) concluded that auxin herbicides, based on phenoxyacetic and propionic acid, were the most susceptible to breakdown by microorganisms of the many pesticides applied to the soil. The importance of soil microorganisms in the breakdown of these herbicides is well known from the work of Audus (1964)(A55) and others. Webster (1967)(A57) has briefly reviewed the literature on the influence of plant growth-regulator auxin herbicides on the host/parasite relationships of nemotodes, in which 2,4-D has been shown to increase nematode reproduction in plant callus cultures. In addition, plant cell hypertrophy and proliferation, which is a common effect of 2,4-D in many plants, provides highly suitable conditions for development of nematodes. In this way, susceptibility of a normally nematode-resistant variety of oats could be induced, although there did not appear to be any greater susceptibility of a non-resistant variety.

In conclusion, the work of Bollen (1961) and many others suggest no significant impact on soil microbes at rates of application used in forest or range spraying.

F. Hazards to Fish and Aquatic Organisms

Under field conditions the toxicity of a pesticide in water is affected by a number of factors in addition to those that affect its performance on land. Thus acidity, hardness of the water, and the sorbent qualities of suspended organic matter in the water may directly effect the toxicity. The trophic nature of the ecosystem, the oxygen status of the water in respect of both producers and demand, and the amount of movement of water both within the system and in terms of flow will affect the concentration of the chemical, its persistence and its possible toxic side effects. Because of these, and many other interacting factors, the toxicity of a given formulation of a given chemical compound to an individual species will vary under field conditions depending upon the nature of the water body and the immediate environment. For this reason, toxicities to fish and aquatic organisms are usually estimated in terms of median tolerance limit for exposure to a given concentration of the pesticide, for a given length of time (TLmx) (A).

In addition to direct or indirect toxicity, the effects on aquatic organisms of the removal of the substrate that gives

them food and shelter must also be considered. For instance, in one of the Tennessee Valley Authority's reservoirs two applications of 2,4-D controlled considerable acreages of Eurasian water milfoil (Myriophyllum spicatum). The eradication of the plant eliminated the substrate that might have been colonized by large populations of epiphytic insects such as the larvae of midges, mayflies, and dragonflies (Smith and Isom 1967) (A69). It has also to be recognized that very heavy infestations of submerged or floating aquatic plants may interfere with the passage of nutrients and considerably reduce the temperatures and dissolved oxygen values of the water (Fish 1966)(A64). Thus, any possible hazards from the use of a herbicide may be outweighed by the advantages gained from the removal of the vegetation.

Reviews of toxicity hazards to fish of a range of pesticides, including auxin herbicides, have been made by Bauer (1961)(A60), Bandt et. al. (1962)(A59), and Cope (1965 and 1966)(A61). Cope (1966)(A61) noted that variations in formulation gave rise to greater differences in toxicity than the differences in toxicity between the basic compounds. Ester formulations were often more toxic than amine or metallic salt formulations. Similar observations were made by Lhoste (1959)(A67) who reviewed effects on a number of crustaceans, aquatic insects, and molluscs.

Trout (Salmo trutta) are normally regarded as being amongst the most sensitive fish to water pollution. Alabaster (1958)(A58) has given median tolerance limits for 24 and 48 hour (TLm24 and TLm 48) exposures of trout to 2,4-D or 2,4,5-T, or to mixtures of these two compounds, of 9.5 to 250 p.p.m., depending on formulation, compared to 1,150 to 2,000 p.p.m. for sodium chlorate or 0.005 p.p.m. for phenyl mercuric acetate. Holden (1964)(A65) devised a formula for comparing the likely toxic hazards to trout from a number of pesticides applied at agricultural rates. The following comparative estimates of hazard were given: aldrin = 70, PCP = 7, MCPA = 1.5, 2,4-D = 1, 2,4,5-T = 0.5, paraquat = 1/12, simazine = 1/27, diquat = 1/40, dalapon = 1/46, TCA = 1/120, and aminotriazole = 1/150.

Perch (Perca fluviatilis) and roach (Rutilus rutilus) are unlikely to be affected by 2,4-D, or 2,4,5-T (Bandt 1957)(A59) at rates of application used for aquatic weed control. In later trials Bandt et. al. (1962)(A59) found threshold values for toxicity to perch and roach of 2,4-D of 75 p.p.m. of 2,4,5-T of 55 to 60 mg/litre and of 2,4-D + 2,4,5-T mixtures of 5 to 12 mg./litre. Davis and Hardcastle (1959)(A68) established median tolerance limits over a 24 hour period (TLm24) for bluegill sunfish (Lepomis macrochirus) to a number of herbicides. Values obtained when the compounds were added to relatively pure water were 2,4-D = 39 p.p.m., MCPA = 20 p.p.m., 2,4-DB = 20 p.p.m., and 2,3,6-TBA = 1,800 p.p.m. Cope (1966)(A61)

noted delays in spawning of bluegill sunfish of up to two weeks after treatment of water with the propylene glycol butyl ether ester of 2,4-D at five and 10 p.p.m. However, no other effects were noted on reproduction or on survival of fry. In pond experiments, death of some fish as a result of 2,4-D treatment led to increased size in the survivors, probably as a result of the greater food supply available to the individual fish. In further trials with bluegill sunfish, Hughes and Davis (1963)(A66) and Davis and Hughes (1963)(A66) reported on effects of different formulations of 2,4-D and other auxins. Their tests showed 2,4-D and 2,4,5-T esters to have TLm24 ranging from 1.8 to 10 p.p.m. depending on the ester used. Dimethylamine salts of 2,4-D and 2,4,5-T had TLm24 of 162 to 542 p.p.m. and 144 p.p.m., respectively, compared to the alkyl amine salt of MCPA of 163.5 p.p.m. and of 2,4-D acid of 8.0 p.p.m. This work (which is referred to in Cope 1966 (A61), see above) shows the wide differences in toxicity that can occur in different formulations and the care which must therefore be taken in assessing the toxicity of an individual product before recommending it for use as an aquatic herbicide.

In addition to work on fish, Walker (1962)(A70) has reported effects on a variety of bottom-feeding fish food organisms following application of 2,4-D to plastic enclosures at 1.0 to 4.0 p.p.m. Lhoste (1959)(A67) has reported that ester formulations of 2,4-D or mixtures of 2,4-D and 2,4,5-T affected crustaceans, aquatic insects, and mollus·s in the range of 0.1 to 3.3 p.p.m.

The results of these various investigations suggest that at herbicidal rates of application of auxins the hazards from acute or chronic toxicities to aquatic organisms are low (B). Nevertheless in some instances the dose rates required for effective herbicidal action for example in estuaries or where the chemical is likely to be rapidly dispersed, may give rise to local and perhaps short term concentrations not far removed from those required for toxic effects on some organisms at susceptible stages of their life history. In such cases, design the application of the phenoxy herbicide to minimize the probability of entry of the chemical to the water.

As may be seen from Table I, it does make a difference which 2,4-D compound is used in aquatic weed control. It is readily apparent that the amine salts are less toxic to these fish than the esters. The effect of 2,4-D on fish-food organisms is shown in Table VI-7. It appears that 1 ppmw of 2,4-D gives about 43% reduction in weight of fish food in one week and about 90% in one year; it should be borne in mind that these data were collected in plastic enclosures and the data may not be strictly comparable to the results expected in field use of this herbicide. Table II presents some data on the effect of herbicides on estuarine organisms including specifically oysters, shrimp, juvenile fish and phytoplankton. This table shows the activities of some other herbicides of interest to this particular report (B). Rawles (1965)(A68) also studies the effect of the 2,4-D

herbicides on caged blue crabs (Callinectes sapidus), eastern oysters (Crassostrea virginica), soft shell clams (Mya arenaria), and various species of fish. Under conditions used to control Eurasian milfoil (Myriophyllum spicatum) only 2,4-D acetamide at 20 lb/acre (ae) was toxic to the test animals; the butyl and isooctyl esters were effective and nontoxic.

TABLE I - EFFECT OF VARIOUS 2,4-D COMPOUNDS ON FISH
(after J. M. Lawrence, 1966)

Compound of 2,4-D	Conc. (ppm)	Species	Time(Hr.)	Remarks
Alkanolamine salt	435-840	Bluegill	48	LD 50
Dimethylamine salt	166-458	Bluegill	48	LD 50
Isooctyl ester	8.8-59.7	Bluegill	48	LD 50
Dimethylamine salt	10	Fathead Minnow	96	LD 50
Acetamide	5	Fathead Minnow	96	LD 50
Oil soluble amine salt	2	Bluegill, Fathead Minnow	4 (Mo)	LD 10
Propylene glycol butyl ether ester	2	Bluegill, Fathead Minnow	4 (Mo)	LD 10
Butoxyethyl ester	2	Bluegill & Fathead	72	LD 70-100
Butyl and isopropyl esters, mixed	1.5-1.7	Bluegill	48	LD 50
N,N-Dimethyl cocoamine salt	1.5	Bluegill	48	LD 50
Ethyl ester	1.4	Bluegill	48	LD 50
Butyl ester	1.3	Bluegill	48	LD 50
Isopropyl ester	1.1	Bluegill	48	LD 50
Duomeen-O-amine salt	0.5	Fathead Minnow Bluegill	4 (Mo)	--

TABLE II AVERAGE NUMBERS OF BOTTOM ORGANISMS PER SQUARE FOOT FOLLOWING APPLICATION OF 2,4-D RANGING FROM ONE TO FOUR PPMW IN SIX PLASTIC ENCLOSURES, 1958-1959

Taxonomic Group	Control	One Week	Six Weeks	12 Months
Mayfly nymphs	4.00	0.17	0.17	--
Horsefly larvae	12.44	4.50	4.50	3.67
Common midges	17.11	4.50	1.50	0.33
Mosquitoes	0.44	0.33	--	--
Phantom midges	3.00	1.00	3.33	0.33
Biting midges	1.22	0.33	0.50	--
Caddis fly larvae	2.78	1.33	0.17	0.33
Damselfly nymphs	0.22	0.17	--	0.67
Water beetles	0.02	--	0.17	3.33
Aquatic worms	24.11	10.00	4.50	1.67
Leeches	0.11	--	--	--
Clams	5.44	--	--	--
Snails	5.67	0.50	--	--
Total numbers	76.56	22.83	14.83	10.33
Total weight	1.299	0.733	0.175	0.127

Source: C. A. Walker, Toxicological effects of herbicide on the fish environment. Missouri University Engineering Extension series 2. Proceedings of the 8th Annual Air and Water Pollution Conference 1962, pp. 17-34.

Studies on the toxicity of 2,4,5-T to fish have been reported by a number of investigators. Hughes and Davis (1963)(A66) have compared the 48-hour median tolerance limit (TLm) of bluegill sunfish to one salt and five ester products of 2,4,5-T:

Compound 2,4,5-T	48-hr TL (ppm, ae[m])
Dimethylamine salt	144.0
Isooctyl ester, supplier A	31.0
Isooctyl ester, supplier A	26.0
Isooctyl ester, supplier B	10.4
Propylene glycol butyl ether ester	17.0
Butoxyethanol ester	1.4

They concluded that 2,4,5-T compounds were in general more toxic than the corresponding 2,4-D products but they were unable to explain the difference observed in the toxicities of the isooctyl esters of 2,4,5-T from different suppliers.

Fish are more susceptible than birds to the butoxyethyl ester of silvex. However, the potassium salt of silvex appears to be less toxic to fish than the ester formulations. No attempt will be made to present all of the fish toxicity data and the reader is referred to the Pesticide Wildlife studies (1963, 1964). Some fish such as the rainbow trout appear at times to be highly resistant to silvex (Cope reports the LD 50 for a 96-hour exposure to be 1,300 ppm) while at other times they appear to be fairly sensitive to silvex

(fish and wildlife report a 96-hour LD 50 of 14.8 ppm). Five out of five fathead minnows were able to survive a 72-hour exposure to 150 ppm of the potassium salt of silvex but other experiments indicate that the safe limit for fathead minnows is between 1 and 3 ppm of the butoxyethanol ester of silvex. Experience with silvex in treated ponds confirms the observation that levels of 3 ppm and above produced liver degeneration lesions, testicular degenerative lesions, atrophy of the spermatic tubules and abnormal spermatozoa on redear sunfish. No comparable changes were seen in the ovaries.

The possible hazard of aquatic weed control procedures to water fowl was considered and analysis of the levels of silvex in the tissues of four ring-necked ducks, six coots, one lesser scaup, one green-winged teal and one gadwall showed low or no detectable residues.(B)

The effect of silvex to possible fish foods has shown that the nymphs of the stonefly (Pteronarcys) could tolerate 5.6 ppm for 24 hours but only 0.32 ppm for 96 hours. Half of the Daphnia magna exposed to 100 ppm of the potassium salt of silvex for 26 hours were immobilized; this is a sign of toxicity but the level is far above the usual 2 ppm used for aquatic weed control. (B)

In summary, the toxicity of silvex is not great to animals, birds and other wildlife; however, there is much variability in the response of fish to silvex and some species may be injured or

killed at levels normally used for aquatic weed control. The potassium salt appears less hazardous to the fish than the butoxyethyl ester. (B)

G. Hazards to Wildlife

Hazards to wildlife from auxin herbicides have been reviewed by Rudd and Genelly (1956)(A72), Springer (1957)(A22), and Mellanby (1967)(A71).

With any material having biological activity a risk of acute or chronic toxicity is always present; however, authenticated incidents of widescale poisoning of wild animals by these herbicides have not been reported.

The real problem from the use of auxin herbicides in regard to wildlife is ecological and not toxicological. The altering of habitat can be a hazard to all forms of wildlife. The size of treatment areas and the intensity of use (frequent applications) become important considerations. Intensity of treatment (repeated applications) is generally associated with agriculture land. This rarely becomes a problem on forest or range lands. The size and location of treatment areas on forest and range land is of utmost importance in considering the effect of spraying on wildlife. Spray areas must be designed to leave sufficient "reservoirs" of habitat.

Fortunately, today there are application techniques and adequate spray equipment at our disposal to leave untreated areas in about any design that is desired.

REFERENCES FOR SECTION VI

A.- J. M. Way, 1969. Toxicity and Hazards to Man, Domestic Animals, and Wildlife From Some Commonly Used Auxin Herbicides. Herbicide Reviews Vol. 26.

A1 Barnes, J. M. Chemicals and Man. Toxic hazards from pesticides used in agriculture. In: Chemicals and the land, p. 135. Proc. Symp. Yorkshire Agr. Soc., Askham Bryan, York, England (1965)

A2 Edwards, C. J., and W. E. Ripper, Droplet size, rates of application and the avoidance of spray drift. Proc. 1st Brit. Weed Control Conf., p. 348 (1953)

A3 Fetisov, M. E., Problems of occupational hygiene in work with herbicides of the 2,4-D group. Gigiena i Sanit. 31, 28 (1966)

A4 Monarca, G., and G. Di Vito, Sull' intossicazione acuta da diserbante (acido 2,4-diclorofenossiacetico). Folia Med. (Naples) 44, 480 (1961)

A5 Nielsen, K. B. Kaempe, and J. Jensen-Holm, Fatal poisoning in man by 2,4-dichlorophenoxyacetic acid (2,4-D), Determination of the agent in forensic materials. Acta Pharmacol. Toxicol. 22, 224 (1965)

A6 Vallet, G., Les intoxications par les herbicides recents. Concours Med. 87, 4633 (1965)

A7 Kephart, L. W., Moderator of session. Proc. 2nd N. Central Weed Control Conf., p. 73 (1945)

A8 Bjorklund, N. E., and K. Erne. Toxicological studies of phenoxyacetic herbicides in animals. Acta Vet. Scand. 7,364 (1966)

A9 Clark, D. E. Young, R. L. Younger, L. M. Hunt, and J. K. McLaran, The fate of 2,4-dichlorophenoxyacetic acid in sheep. J. Agr. Food Chem. 12, 43 (1964)

A10 Dalgaard-Mikkelsen, S. V., and E. Poulsen. Toxicology of herbicides. Pharmacol. Rev. 14, 225 (1962)

A11 Dobson, N. Chemical sprays and poultry. Agriculture (London) 61,415 (1954)

A12 Dunachie, J. F., and W. W. Flethcher. Effect of some herbicides on the hatching rate of hen's eggs. Nature 215, 1406 (1967)

A13 Erne, K. Distribution and elimination of chlorinated phenoxy-acetic acids in animals. Acta Vet. Scand. 7, 240 (1966a)

A14 Goldstein, H. E., and J. F. Long. Observations on cattle, sheep, and swine exposed to 2,4-D, 2,4,5-T and Dalapon herbicides. Proc. 13th S. Weed Control Conf. p. 5 (1960)

A15 Gutenmann, W. H. and D. J. Lisk. Conversion of 4-(2,4-DB) herbicide to 2,4-D by bluegills. N.Y. Fish Game J. 12, 108 (1965)

A16 Khanna, S., and S. C. Fang. Metabolism of C^{14}-labeled 2,4-dichlorophenoxyacetic acid in rats. J. Agr. Food Chem. 14,500 (1966)

A17 Lisk, D J. How now brown cow? Farm Research 32, 15 (1966)

A18 Palmer, J. S. Chronic toxicity of 2,4-D alkanolamine salts to cattle. J. Amer. Vet. Assoc. 143,398 (1963)

A19 Roberts, R. E., and B. J. Rogers. The effect of 2,4,5-T brush spray on turkeys. Poultry Sco., 36 703 (1957)

A20 Rowe, V. K., and T. A. Hymas. Summary of toxicological information on 2,4-D and 2,4,5-T type herbicides and an evaluation of the hazards to livestock associated with their use. Amer. J. Vet. Research 15, 622 (1954)

A21 Strach, S., and M. Botiosiewicz. Badania nad toksycznoscia chwastobojezego preparatu "Pielik" dla swin. Med. Weterynar. (Poland) 20, 662 (1964)

A22 Springer, P. F. Effects of herbicides and fungicides on wildlife N. Carolina Pesticide Manual, p. 88 (1957)

A23 Bache, C. A., D. J. Lisk, D. G. Wagner, and R. G. Warner. Elimination of 2-methyl-4-chlorophenoxyacetic acid and 4-(2 methyl-4-chlorophenoxybutryic) acid in the urine from cows. J. Dairy Sci. 47, 93 (1964)

A24 Beevers, L., and R. H. Hageman. Effects of 2,4-D application on nitrate metabolism in higher plants. Plant Physiol. 37, suppl. XXIII (1962)

A25 Berg, R. T., and L. W. McElroy. Effect of 2,4-D on the nitrate content of forage crops and weeds. Can. J. Agr. Sci., 33, 354 (1953)

A26 Buck, W. B., W. Binns, L. James, and M. C. Williams. Results of feeding of herbicide-treated plants to calves and sheep. J. Amer. Vet. Med. Assoc. 138, 320 (1961)

A27 Case, A. A. Some aspects of nitrate intoxication in livestock. J. Amer. Vet. Med. Assoc., 130,323 (1957)

A28 Davidson, W. B., J. L. Doughty, and J. L. Bolton. Nitrate poisoning of livestock. Can. J. Comp. Med. Vet. Sci. 5, 303 (1941)

A29 Fertig, S. N. Livestock poisoning from herbicide treated vegetation. Proc. 6th NE, Weed Control Conf., p. 13 (1952). Herbicidal poisoning of livestock. Proc. 7th NE. Weed Control Conf. Suppl. 44 (1953)

A30 Frank, P. A., and B. H. Grigsby. Effects of herbicidal sprays on nitrate accumulation in certain weed species. Weeds, 4, 206 (1957)

A31 Grigsby, B. H. and C. D. Ball. Some effects of herbicidal sprays on the hydrocyanic acid content of leaves of wild black cherry (Prunus serotina Ehr.) Proc. 6th NE Weed Control Conf. p. 327 (1952)

A32 Gilbert, C. S., H. F. Eppson, W. B. Bradley, and O. A. Beath. Nitrate accumulation in cultivated plants and weeds. Wyoming Univ. Agr. Expt. Sta. Bull. No. 277 (1946)

A33 Lynn, G. E. and R. C. Barrons. The hydrocyanic (HCN) content of wild cherry leaves sprayed with a brush killer containing low volatile esters of 2,4-D and 2,4,5-T. Proc. 6th NE. Weed Control Conf. p. 331 (1952)

A34 Phillips, W. E. J. Metabolism of carotene and vitamin A following ingestion of the herbicide MCPA. Can. J. Animal Sci. 44, 29 (1964)

A35 Savage, A. Nitrate poisoning from sugar beet tops. Can. J. Comp. Med. Vet. Sci. 13, 9 (1949)

A36 Stahler, L. M. and E. I. Whitehead. The effect of 2,4-D on potassium nitrate levels in leaves of sugar beets. Science 112, 749 (1950)

A37 Sund, J. M., M. J. Wright, and J. Simon. Nitrates in weeds cause abortion in cattle. Proc. 17th N. Central Weed Control Conf., p. 31 (1960)

A38 Swanson, C. R. and W. C. Shaw. The effect of 2,4-dichlorophenoxy-acetic acid on the hydrocyanic acid and nitrate content of sudan grass. Agron. J. 46, 418 (1954)

A39 Whitehead, E. I., J. Kersten, and D. Jacobsen. The effect of 2,4-D spray on the nitrate content of sugar beet and mustard plants. Proc. S. Dakota Acad. Sci. 35, 106 (1956)

A40 Willard, C. J. Indirect effects of herbicides. Proc. 7th N. Central Weed Control Conf. p. 110 (1950)

A41 Williams, M. C., and E. H. Cronin. Effect of silvex and 2,4,5-T on alkaloid.

A42 Bradley, W. B., H. F. Eppson, and O. A. Beath. Livestock poisoning by oat hay and other plants containing nitrates. Wyoming Agr. Expt. Sta. Bull. No. 241, (1940)

A43 Adams, J. B. Effects of spraying 2,4-D amine on coccinellid larvae. Can. J. Zool. 38, 285 (1960)

A44 Antoine, O. Les antiparasitaires et les animaux domestiques, risques pour les poissons, les abeilles et la vie sauvage. Parasitica (Gembloux) 22, 107 (1966)

A45 Byrdy, St. Untersuchungen uber die Wirkung des 2,4-D-Praparates "Pielik" auf Bienen, Tagungsbericht 2. Inter. Arbeitstagung der Arbeitsgemeinschaft. Toxicologie von Pflanzensch. Mitt Berlin 54, 15 (1962)

A46 Glynne Jones, G. D. and J. U. Connell. Studies of the toxicity to worker honeybees (Apis mellifera L.) of certain chemicals used in plant protection. Ann. Applied Biol. 41, 271 (1954)

A47 Ishii, S., and C. Hirano. Growth responses of larvae of the ricestem borer to rice plants treated with 2,4-D. Entomol. Expt. Appl. 6, 257 (1963)

A48 Johansen, C. Bee poisoning. A hazard of applying agricultural chemicals. Wash. State Coll. Agr. Expt. Sta. Circ. No. 356 (1959)

A49 King, C. C. Translocation of C^{14}-2,4-D and C^{14}-amitrole or their metabolites to nectar in plants. Research Rept. 17th N. Central Weed Control Conf. p. 105 (1960a)

A50 Maxwell, R. C., and R. F. Harwood. Increased reproduction of pea aphids on broad beans treated with 2,4-D. Ann. Entomol. Soc. Amer. 53, 199 (1960)

A51 Palmer-Jones, T. Effect on honey-bees of some chemical weedkillers. New Zealand J. Agr. Research 3, 485 (1960)

A52 Robinson, A. G. Note on fecundity of the pea aphid Acrythosiphon pisum caged on plants of broad bean vicia faba L., treated with various plant growth-regulators. Can. Entomologist 91, 527 (1959)

A53 Wahlin, B. Bina och hormonderivaten. Vaxtskyddsnotiser, Stockholm 14, 45 (1950)

A54 Haragsimova, L. Einfluss der in der Tschechoslowakei im Pflanzenschutz gebrauchlichen chemischen Mittel auf die Honigbiene (Apis mellifera L.) Tagungsbericht 2. Inter. Arbeitstagung der Arbeitsgemeinschaft. Toxicologie von Pflanzensch, Mitt. Berlin 54, 35 (1962)

A55 Audus, L, J. Herbicide behavior in the soil. In: The Physiology and biochemistry of herbicides (Ed., L. J. Audus). London-New York Academic Press (1964)

A56 Bollen W. B. Interactions between pesticides and soil micro-organisms. Ann. Rev. Microbiol. 15, 69 (1961)

A57 Webster, J. M. Some effects of 2,4-dichlorophenoxyacetic acid herbicides on nematode-infested cereals. Plant Pathol. 16, 23 (1967)

A58 Alabaster, J. S. Toxicity of weedkillers, algicides and fungicides to trout. Proc. 4th Brit. Weed Control Conf., p. 84 (1958)

A59 Bandt, H. J. Uber die Giftwirkund von Herbiziden auf Fische. Z. Fischerei 6, 121 (1957)

A60 Bauer, K. Studren uber Nebenwirkungen von Pflanzenschutzmitteln auf Fische und Fischnahrtiere. Mitt. biol. Bundesanstalt Land u Forstwirtsch, Berlin-Dahlem 105, 5 (1961)

A61 Cope, O. B. Some responses of fresh-water fish to herbicides. Proc. 18th S. Weed Control Conf. p. 439 (1965)

A62 Davis, B. N. K. The immediate and long-term effects of the herbicide MCPA on soil arthropods. Bull. Entomol. Research 56, 357 (1965)

A63 Davis, J. T., and W. S. Hardcastle. Biological assay of herbicides for fish toxicity. Weeds 7, 397 (1959)

A64 Fish, G. R. Some effects of the destruction of aquatic weeds in Lake Rotoiti, New Zealand. Weed Research 6, 350 (1966)

A65 Holden, A. V. The possible effects on fish of chemicals used in Agriculture. J. Inst. Sewage Purif. 4, 361 (1964)

A 66 Hughes, J. S., and J. T. Davis. Variations in toxicity to bluegill sunfish of phenoxy herbicides. Weeds 11, 50 (1963)

A67 Lhoste, J. Les repercussions de l'emploi des desherbants chimiques sur la faune aquatique C. r. Reun. Tech. d'Athenes de I'UICN 4, 253 (1959)

A68 Rawls, C. K. Field tests of herbicide toxicity to certain estuarine animals. Chesapeake Sci. 6, 150 (1965)

A69 Smith, G. E., and B. G. Isom. Investigation of effects of large-scale applications of 2,4-D on aquatic fauna and water quality. Pesticides Monitoring J. 1, 16 (1967)

A70 Walker, C. R. Toxicological effects of herbicides on the fish environment. Proc. 8th Air Water Pollution Abatement Conf. p. 17, (1962)

A71 Mellanby, K. Pesticides and pollution. New Naturalist No. 50 London-Glasgow. Collins (1967)

A72 Rudd, R. L., and R. E. Genelly. Pesticides. Their use and toxicity in relation to wildlife. Calif. Fish Game Bull. No. 7 (1956)

B. House W. B., L. H. Goodson, H. M. Gadberry and K. W. Docktur, 1967. Assessment of ecological effects of extensive or repeated use of herbicides. Midwest Res. Inst. Proj. 3103-B. Adv. Res. Proj. Agency order 1086 Dept. of Def. Contract No. DAHC15-68-C-0119, 369 p.

B1 Palmer, J. S. Chronic toxicity of 2,4-D alkanolamine salts to cattle. J. Amer. Vet. Med. Assoc. 1963.

B2 Clark, D. E., J. E. Young, R. L. Younger, L. M. Hunt, and J. K. McLaren, 1964. The fate of 2,4-dichlorophenoxyacetic acid in sheep. J. Agr. Food Chem. 12: 43-45.

B3 Khanna, Suchitra, and S. C. Fang. 1966. Metabolism of C^{14}-labeled 2,4-dichlorophenoxyacetic acid in rats. J. Agr. Food Chem. 14: 500-503

B4 Buck, W. B. et. al. 1961. Results of feeding herbicide treated plants to calves and sheep. J. Amer. Vet. Med. Assoc. 138, 320

B5 Stahler, L. M. and E. I. Whitehead, 1950. The effect of 2,4-D on potassium nitrate levels in leaves of sugar beets. Science 112, 749.

B6 Lynn, G. E. and K. C. Barrons, 1952. The hydrocyanic (HCN) content of wild cherry leaves sprayed with a brush killer containing low volatile esters of 2,4-D and 2,4,5-T. Proc. 6th NE Weed Control Conf. p. 331.

C. Report of the Advisory Committee on 2,4,5-T. 1971. To the administrator of the Environment Protection Agency.

C1 Sparschu, G. L., F. L. Dunn, R. W. Lisowe and V. K. Rowe, 1971. Study of the effects of high levels of 2,4,5-Trichlorophenoxy-acetic acid (2,4,5-T) on rat fetal development. Unpublished study.

C2 Johnson, J. E. 1970. The public health implications of widespread use of the phenoxy herbicides and picloram. Presented at the Symposium on Possible Public Health Implications of widespread use of pesticides, American Institute of Biological Sciences, Bloomington, Indiana, August 26, 1970.

C3 King. C. T. G., 1971. Teratogenicity studies of 2,4,5-T and 2,4-D Unpublished report, February 25, 1971.

C4 Tschirley, F. H., 1971. Report on status of knowledge regarding 2,4,5-T. Submitted by the USDA to the EPA, March 5, 1971. 2,4,5-T Advisory Committee AE-20

C5 2,4,5-T Advisory Committee Exhibits I-13a, I-14, and I-15

D. St. John, L. E. and D. J. Lisk, 1964. Fate of atrazine, kuron, silvex, and 2,4,5-T in the dairy cow. Journal of Dairy Sciences. November 1964. Vol. XLVII. No. 11, pp. 1267-1270

REPORT

ON

BACKGROUND INFORMATION

FOR

SIMAZINE

2-23-72
Lisle R. Green

An Outline of Background Information for the Herbicide SIMAZINE

I. General Information

A. Common name

1. Simazine

B. Chemical name

1. 2-chloro-4,6-bis(ethylamino)-s-triazine

C. Registered uses[1/]

1. As a selective herbicide to control the germinating seedlings of most annual broadleaf and grassy weeds in:

a. Field and forage crops

(1) Alfalfa[2/]

(2) Forage bermudagrasses[2/]

(3) Corn

(4) Sugarcane

(5) Grasses grown for seed (Pacific Northwest only); various perennial grasses used for lawns and turf.

1/ Special precautionary recommendations and statements warning of isolated effects or special problems which may be encountered are published in the sample label and general information brochure published by Geigy Agricultural Chemicals.

2/ Don't graze livestock (sheep, dairy, or beef cattle) for 30 days following a 1 to 3 lb. application, or 60 days following 4 lbs. Don't cut for hay for 60 days following a 1 to 3 lb. per acre application, or 90 days following 4 lbs.

b. Fruit and nut crops

(1) Fruit crops--apples, pears, peaches, grapes, cherries, plums, avocados

(2) Nut crops--walnuts, filberts (in Washington and Oregon)

(3) Bush fruits--blackberries, boysenberries, loganberries, blueberries

(4) Citrus fruits--oranges, lemons, grapefruit

c. Nurseries, Christmas trees, plantings and shelterbelts

(1) Species

American elm
Austrian pine
Arborvitae
Balsam fir
Barberry
Blue spruce
Boxelder
Bush honeysuckle
Caragana
Cotoneaster
Dogwood
Douglas-fir
Fraser fir
Hemlock
Honey locust
Juniper
Mugho pine
Norway spruce
Oregon grape (Mahonia spp.)
Red cedar
Red oak
Red pine (Norway pine)
Red spruce
Russian olive
Scotch pine
Siberian elm
White cedar
White pine
White spruce
Yew (Taxus spp.)

d. Turf grasses for sod

(1) Species

St. Augustine
Centipede
Zoysia grass
Perennial ryegrass
Bentgrasses
Orchardgrass
Tall fescue
Fine fescues

e. Vegetable crops

Asparagus (established)
Artichokes

f. Hard-to-kill perennial weeds such as bull thistle, bindweed, and perennial grasses.

2. Nonselective weed control for noncrop land.

a. At higher rates, simazine is used as a sterilant to remove most or all vegetation from industrial sites, fence rows, railroads, around utility poles, and along roads.

3. Aquatic plants.

An experimental label has been granted for use in aquatic environments for weed and algae control.

D. Formulations manufactured

1. A wettable powder containing 80 percent active ingredient, marketed as Princep 80W.

2. A granular form containing 4 percent active ingredient, marketed as Princep-4G.

E. Dilutions of formulations for use in:

1. Use enough water with the wettable powder to assure thorough, uniform coverage on the soil surface.

a. Ground applications

(1) 20 to 100 gallons of water per acre.

b. Broadcast aerial applications

(1) A minimum of 1 gallon of water for each 1 lb. of simazine to be applied per acre for preemergence applications, up to 15 gallons per acre.

F. Rate and method of application

1. Rate of application.

a. Selective weed control to eliminate annual grasses and broadleaf annual weeds from perennial vegetation.

(1)	Sand & loamy sand, low OM	Do not use
(2)	Fine sand & sandy loam	1 to 2 1/2 lb./acre
(3)	Loam & clay loam, low OM	2 1/2 to 3 lb./acre
(4)	Clay, or other soils high in OM	3 to 4 lb./acre
(5)	High organic clays	4 to 5 lb./acre

b. Nonselective weed control on noncropland.

(1)	Most annual and many perennial broadleaf and grass weeds	12 1/2 to 25 lb./acre
(2)	For "sterilant" effect of about 3 to 4 years, depending on rainfall.	25 to 50 lb./acre

c. Water plants.

(1)	Submerged, in ponds	0.5 to 2 ppmw
(2)	Sensitive emergent [bulrush (*Scirpus*)], *Carex*, *Polygonum*, Needlerush (*Eleocharis*), arrowhead (*Saggittaria*), willow (*Salix*) [Walker 1964]	10 to 20 lb./acre

(F. - d. Discussion)

There are numerous reports in the literature which indicate that soil organic matter is the most active soil component in adsorbing simazine, thus reducing its phytotoxicity and requiring larger applications to do the same weed control job. Many of these references are included in a review article by Hayes (1970). There are also numerous references in Residue Reviews 32 (1970) which indicate that clay content of soil is also important, and some where clay content didn't seem important. Type of clay is important--montmorillonite being more active than kaolinite, for example (Weber 1970). Perhaps an "average" of the clay-organic matter situation was obtained by results of Nearpass (1965) who found adsorption of simazine to be significantly correlated with percent of clay and highly significantly correlated with organic matter and titratable acidity in 18 soils.

Evans et al. (1969) found control of downy brome with simazine at 1 pound per acre averaged about 73 percent. There was good broadleaf weed control but no control of Russian thistle. Green and Benedict (unpublished manuscript) controlled downy brome and other annuals with simazine at 1 1/2 to 3 pounds per acre on sandy loam soils.

Simazine at 2 pounds per acre controlled hoary alyssum (Berteroa incana (L.) D.C.), a perennial weed, in alfalfa (Kust 1969). Simazine at 1 1/2 pounds per acre controlled annuals for one year in pecan orchards and at 4 pounds, gave nearly complete control of all weeds, including

nutsedge and bermudagrass. The soil was a loamy fine sand (Norton and Storey 1970). Simazine at 10 pounds per acre caused visible foliar injury but no height or weight loss to young Japanese maple trees, and there was no injury to yew (Danielson and May 1969). There is lots of experience which demonstrates the safety of older trees when simazine is applied at 1 to 4 pounds per acre. There is some indication that toxicity of simazine has decreased as soil moisture decreased (Grover 1966, Buchholtz 1965, and Evans et al. 1969). "Holdover" effects are generally small at less than 2 pounds of simazine per acre, but sensitive plants are damaged at 2 pounds or more. At 10 pounds or more, residual effects can be expected for at least three years. Most residual simazine is in the surface few inches of soil.

Leaching has occurred to greater depths with 4 pounds than with 2 pounds, and deeper when rainfall was concentrated rather than spread over several smaller storms (Rodgers 1968).

2. Method of application

   a. Ground sprayers--Most low pressure (25 to 40 lb. pressure) sprayers can be used. Teejet 8003 or 8004 fan-type nozzles or equivalent. Tank must have mechanical or bypass agitation.

   b. Aerial spray.

   c. Broadcasting of pellets.

      (1) Cyclone type hand spreaders

      (2) Field spreaders

      (3) Aerial

3. General

   a. Application of either spray or granules should be made prior to weed emergence, and certainly before weeds are more than an inch or so tall. If taller than this, amitrole or other herbicide that works through the foliage should be applied with simazine.

   b. Simazine has little or no foliar activity, and requires rain or irrigation to take it into the root zone for absorption.

G. Tolerances in food or feed and other safety limitations

1. The Federal Food and Drug Administration has set tolerances for residues and simazine on certain raw agricultural commodities as follows:

   a. 15.00 ppm in or on alfalfa, bermudagrass, other grass.

   b. 10.00 ppm in or on asparagus.

   c. 0.50 ppm in or on artichokes.

d. 0.25 ppm in or on almonds (hulls and nuts), apples, avocados, blackberries, blueberries, boysenberries, cherries, fresh corn including sweet corn (kernels plus cobs with husks removed), corn grain (including popcorn), corn forage or fodder (including field corn, sweet corn, and popcorn), cranberries, currants, dewberries, grapefruit, grapes, lemons, loganberries, macadamia nuts, olives, oranges, peaches, pears, plums, raspberries, strawberries, walnuts.

0.02 ppm (negligible residue) in eggs, milk, meat, fat, and meat by-products of cattle, goats, hogs, horses, poultry, and sheep.

2. Consult the Federal Food and Drug Administration for changes and additions. These will also be reflected in the most recently issued Geigy Agricultural Chemical Company technical bulletin or labels covering simazine.

3. The marketing of raw agricultural commodities having residues in excess of their permitted tolerances, or marketing those for which no tolerances have been set and bearing residues, will violate Federal Law when shipped in interstate commerce and may violate State Law.

H. Manufacturer

1. Geigy Agricultural Chemicals
Saw Mill River Road
Ardsley, New York 10702

II. Toxicity data on formulation to be used

A. Safety data

1. Acute mammalian studies

a. Oral

Available evidence and experience indicates that simazine has low toxicity to animals, and most likely to man also. The acute oral toxicity (LD_{50}) of simazine to rats, mice, rabbits, chickens, and pigeons is in excess of 5,000 mg (5g)/kilogram (kg) of body weight (Geigy Agricultural Chemical Co. 1970).

Cattle fed 250 mg of simazine/kg of body weight as a drench showed poisoning symptoms after one dose, but survived 3 doses with 11 percent weight loss (Palmer and Radeliff 1969).

No cases of poisoning in man have been reported from ingestion of simazine.

b. Dermal

The acute dermal LD_{50} of simazine to albino rabbits is greater than 10g/kg. In a 21-day repeated dermal study on albino rabbits, the LD_{50} was 2g/kg (Geigy Agricultural Chemical Co. 1970).

No substantial skin irritation has been reported from either experimental or commercial use.

c. Inhalation

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/l of atmosphere.

d. Eye and skin irritation

No serious skin or eye irritation has been reported for experimental or commercial use.

2. Subacute studies

a. Oral, b. Dermal, c. Inhalation

Two year chronic oral feeding studies, in which male and female rats were given daily dosages at various rates as high as 100 ppm of simazine 50W in the diet, resulted in no gross or microscopic signs of systematic toxicity due to ingestion (Geigy Agricultural Chemical Co. 1970).

Two yearling cattle showed visible poisoning symptoms after 3 and 10 doses of 25 mg/kg. There were no symptoms at 10 mg/kg. One sheep was poisoned at 50 mg/kg after 17 doses, and died after 31 doses, whereas another was poisoned with 10 doses but survived. Chickens dosed at 50 mg simazine/kg showed reduced weight gain. Application rates in excess of 3 pounds per acre would be hazardous for grazing cattle and in excess of 5 pounds for sheep. The 9.6-pound rate would be hazardous for chickens (Palmer and Radeliff 1969. Sheep fed up to 25 mg/kg for 5 weeks remained normal (Geigy Agricultural Chemical Co. 1970).

3. Other studies which may be required

a. Neurotoxicity

b. Teratogenicity

c. Effects on reproduction

d. Synergism

e. Potentiation

f. Metabolism and mode of action

Simazine enters weeds mainly through the roots. Its most efficient use requires application before weeds germinate, and rainfall sufficient to carry it to the root zone. It is translated through the xylem to the leaves where it disrupts the photosynthetic process (Geigy Agricultural Chemical Co. 1970). Simazine at 0.12 to 1 ppmw inhibited oxygen production through reduction of photosynthesis of aquatic plants (Sutton et al. 1969). It was noted during another study that chlorophyll and cell chloroplast protein was reduced in oat plants subjected to simazine at 1 ppm for 6 days.

g. Avian and fish toxicity

(1) Fish toxicity

Simazine at 3 ppmw was reported to be nontoxic to fish (Flanagan, Proc. NE Weed Control Conf. 14: 502-505), although in another situation, simazine at 2 ppmw killed adult, but not young, redear sunfish in one pond and not in a second (Snow, Proc. So. Weed Control Conf. 16: 329-335). Green sunfish were exposed to simazine by feeding of 3 to 10 mg/kg and by water bath (1 and 3 ppm) from which they absorbed simazine in direct proportion to its concentration in the water. No simazine residue was detected 7 days after either treatment and there appeared to be no damage to the fish (Rodgers 1970). In another study, simazine at 2 ppmw controlled 80 percent of water plants while giving a safety factor for aquatic life. The LD_{50} toxic dose to bottom dwelling organisms was 28 ppmw. LD_{10} values for three sunfish species were 20 ppmw and LD_{50} about 35 (Walker 1964).

(2) Avian toxicity

h. Carcinogenicity

B. Physical-chemical properties

1. Melting point

   a. 225-227° C (437-441° F)

2. Flash point

3. Physical state and color

   a. A noncombustible, white crystalline substance. The commercial product is prepared as a powder or as granules.

4. Density

   a. Molecular weight is 201.7

5. Vapor pressure

a. Temp° C	MM Hg.
10	9.2×10^{-10}
20	6.1×10^{-9}
30	3.6×10^{-8}
50	9.0×10^{-7}

6. Solubility

a. Solvent	Temp° C	Solubility ppm
Water	0	2.0
Water	20 (68° F)	5.0
Water	85	84.0
Methanol	20	400.0
Petroleum ether	20	2.0

5. Stability

Simazine has practically unlimited stability at room temperature within a pH range of 3 to 10 (Geigy Agricultural Chemical Co. 1970).

Several workers have demonstrated photodecomposition in the laboratory after exposure to ultraviolet light, and also under sunlight when simazine was exposed on the soil surface during the summer. In one case, this amounted to 25 percent of its phototoxic effect in 25 days (Jordan et al. 1970).

Volatilization is most likely a source of loss from the soil under conditions of high soil temperature, although simazine is less volatile than most other s-triazines (Kearney et al. 1964) with virtually no volatility of simazine between 25 to 45° C (77 - 112° F). Several investigators have shown simazine losses by volatilization at temperatures from 112° to 212° F (Jordan et al. 1970). Loss from this source is probably very small.

Inactivation of simazine in the field was shown by Talbert and Fletchall (1964) to be greatest when environment was most favorable for growth of microorganisms. The available evidence indicates that slow microbiological decomposition is the principle process involved in dissipation of simazine (Burnside et al. 1961, Ragab and McCollum 1961, Weed Research 1: 131-141, and Proc., British Weed Control Conf. 5: 91-97). The degradation processes are reviewed by Kaufman and Kearney (1970).

III. Efficacy data under field and laboratory conditions

A. Effectiveness for intended purpose when used as directed

Simazine when used at prescribed rates for prescribed crops and conditions seems to perform as advertised. It is registered for and used as a selective herbicide on many perennial crops. It is also registered for nonselective weed control on noncroplands.

B. Phytotoxicity

Simazine is toxic to a wide variety of grassy and broadleaf weeds. It can be used as a selective herbicide because it is relatively resistant to leaching (Ashton 1961, Montgomery and Freed 1959, Roadhouse and Birk 1961, and Rogers 1962), and can readily be placed in the root zone of recently germinated plants. Higher rates have tended to leach deeper than low rates, and leaching is deeper in sandy soil of low organic content than in organic or clay soils, hence deeper rooted perennial plants can be damaged by high rates, or if growing in sandy soils, or if heavy precipitation carries the herbicide into their root zone.

There is variable tolerance among plants to simazine. Corn evidently converts simazine to nontoxic materials (Montgomery and Freed 1961). Western wheatgrass, crested wheatgrass, blue grama, and sedge were less tolerant to s-triazines (simazine) than needle- and - thread and sand dropseed in western Nebraska (Wicks et al. 1965).

C. Translocation

Simazine is absorbed through plant roots with little or no foliar penetration. It is translocated through the xylem, and accumulates in the apical meristems and leaves.

D. Persistence in soil, water, or plants

1. Persistence in soil

Simazine will tend to persist longer in fine textured than in sandy soils, in arid more than moist situations, in cold more than warm soils, and in situations otherwise not conducive to chemical and microorganism action.

The residual activity of simazine in soil at selective rates for the specific soil types is such that many rotational crops can be planted one year after application. However, there is frequently some simazine residue that may affect sensitive crops (Herbicide Handbook 1967, Lewis and Lilly 1966, Buchholtz 1965, and Burnside et al. 1965).

Under arid conditions persisting near Reno, Nevada, simazine at 1 pound per acre controlled annual weeds if rainfall was normal, and perennial grasses could be planted a year after the simazine treatment (Evans et al. 1969). Green and Benedict (unpublished manuscript) found simazine at 3 pounds per acre restricting downy brome growth for a year, with partial downy brome recovery in two years.

E. Compatibility with other chemicals

Simazine is compatible with most other herbicides and fertilizers at normal rates. It is frequently used with amitrole or other foliar absorbed herbicide if weeds are already growing actively.

IV. Environmental impact

A. Effects on non-target organisms

These are believed to be small.

B.

Literature Cited

Ashton, F. M. 1961. Movement of herbicides in soil with simulated furrow irrigation. Weeds 9: 612-619.

Buchholtz, K. P. 1965. Factors influencing oat injury from triazine residues in soil. Weeds 13(4): 362-367.

Burnside, O. C., E. L. Schmidt, and R. Behrens. 1961. Dissipation of simazine from the soil. Weeds 9(3): 477-484.

Burnside, O. C., G. A. Wicks, and C. R. Fenster. 1965. Herbicide longevity in Nebraska soils. Weeds 13(3): 277-278.

Danielson, L. L. and C. May. 1969. Effects of several herbicides on yews and Japanese maples. Weed Science 17(2): 142-144.

Evans, R. A., R. E. Eckert, Jr., B. L. Kay, and J. A. Young. 1969. Downy brome control by soil-active herbicides for revegetation of rangelands. Weed Science 17(2): 166-169.

Flanagan. Proc. NE Weed Control Conf. 14: 502-505.

Geigy Agricultural Chemicals. 1970. Princep herbicide. Tech. Bull. Geigy Chemical Corp., Ardsley, New York. 8 p.

Green, L. R. and E. W. Benedict. Soil sterilants to control herbaceous vegetation on Intermountain fuel-breaks (unpublished manuscript).

Grover, R. 1966. Influence of organic matter, texture, and available water on the toxicity of simazine in soil. Weeds 14(2): 148-151.

Gunther, F. A. and J. D. Gunther. (Editors). 1970. Residue Reviews, Vol. 32. The Triazine Herbicides. Springer-Verlag, New York. 413 p.

Hayes, M. H. B. 1970. Adsorption of triazine herbicides on soil organic matter, including a short review on soil organic matter chemistry. In Residue Reviews 32: 131-174.

Jordan, L. S., W. J. Farmer, J. R. Goodin, and B. E. Day. 1970. Non-biological detoxication of the s-triazine herbicides. In Residue Reviews 32: 267-286.

Kaufman, D. D. and P. C. Kearney. 1970. Microbial degradation of s-triazine herbicides. In Residue Reviews 32: 235-265.

Kearney, P. C., T. J. Sheets, and J. W. Smith. 1964. Volatility of seven s-triazines. Weeds 12(2): 83-87.

Kust, C. A. 1969. Selective control of hoary alyssum in alfalfa. Weed Science 17(1): 99-101.

Lewis, W. M. and J. P. Lilly. 1966. Retreatments of preemergence and post emergence crabgrass herbicides and their residual effects on the establishment of three turfgrasses. Proc., Southern Weed Control Conf. 19: 161-167.

Montgomery, M. and V. H. Freed. 1959. A comparison of the leaching behavior of simazine and atrazine in Chehalis sandy loam. Res. Progress Report, Western Weed Control Conf.

Montgomery, M. and V. H. Freed. 1961. The uptake, translocation and metabolism of simazine and atrazine by corn plants. Weeds 9: 231-237.

Nearpass, D. C. 1965. Effects of soil acidity on the adsorption, penetration, and persistence of simazine. Weeds 13(4): 341-346.

Norton, J. A. and J. B. Storey. 1970. Effect of herbicides on weed control and growth of pecan trees. Weed Science 18(4): 522-524.

Palmer, J. S. and R. D. Radeliff. 1969. The toxicity of some organic herbicides to cattle, sheep, and chickens. Production Report No. 106, USDA Agricultural Research Service, Washington, D. C. 26 p.

Proceedings, British Weed Control Conf. 5: 91-97.

Ragab, M. T. H. and J. P. McCollum. 1961. Degradation of C^{14}-labeled simazine by plants and soil microorganisms. Weeds 9(1): 72-84.

Roadhouse, F. E. B. and L. A. Birk. 1961. Penetration of and persistence in soil of the herbicide 2-chloro-4,6-bis(ethylamino-s-triazine (simazine). Can. J. Plant Sci. 41: 252-260.

Rodgers, C. A. 1970. Uptake and elimination of simazine by green sunfish (Lepomis cyanellus Raf.). Weed Science 18(1): 134-136.

Rodgers, E. G. 1962. Leaching of four triazines in three soils as influenced by varying frequencies and rates of simulated rainfall. Proc. Southern Weed Control Conf. 15: 268.

Rodgers, E. G. 1968. Leaching of seven s-triazines. Weed Science 16(2): 117-120.

Snow. Proc. Southern Weed Control Conf. 16: 329-335.

Sutton, D. L., D. A. Durham, S. W. Bingham, and C. L. Foy. 1969. Influence of simazine on apparent photosynthesis of aquatic plants and herbicide residue removal from water. Weed Science 17(1): 56-59.

Talbert, R. E. and O. H. Fletchall. 1964. Inactivation of simazine and atrazine in the field. Weeds 12(1): 33-37.

Walker, C. R. 1964. Simazine and other s-triazine compounds as aquatic herbicides in fish habitats. Weeds 12(2): 134-139.

Weber, J. B. 1970. Mechanisms of adsorption of s-triazines by clay colloids and factors affecting plant availability. In Residue Reviews 32: 93-130.

Weed Research 1: 131-141 .

Weed Society of America. Herbicide Handbook of the Weed Society of America. W. F. Humphrey Press, Geneva, N. Y. 293 p.

Wicks, G. A., C. R. Fenster, and O. C. Burnside. 1965. Chemical control of downy brome in grasslands of western Nebraska. Weeds 13(3): 202-205.