1,3-DICHLOROPROPENE

a profile

Dow AgroSciences LLC
9330 Zionsville Road
Indianapolis, IN 46268

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Dow AgroSciences

1,3-Dichloropropene SOIL FUMIGANT

Product Types

1,3-Dichloropropene
1,3-Dichloropropene + chloropicrin

Approval Crops U.S.

Over 120 crops including:
Vegetable crops
field crops
nursery crops
planting sites for citrus trees,
deciduous fruit trees,
nut trees and berry bushes and vines

Biological Activity

Major Pests Controlled:

- Plant parasites and nematodes including; burrowing, citrus, cyst (sugar beet, soybean, carrot and wheat), dagger, lance, reniform, ring, rootknot, root lesion (meadow), spiral, sting and stubby root
- garden centipedes (symphylans) wireworms

Suppresses-sugar beet Rhizomania disease, Granville wilt of tobacco, *Fusarium* wilt of cotton, *Verticillium* wilt of mint and potatoes, and aids in the control of bacterial canker in peaches.

PROPERTIES

Environmental Fate

Soil
Aerobic half-life: 1.7 – 53 days
Anaerobic half-life: 2.4 – 9.1 days
K_{oc}: 20-43

Water
Solubility: 2.2 g/L
Hydrolysis half-life:
3 days at 30°C – 51 days at 10°C

Air
Photolysis half-life: 7 – 12 hours
Vapor Pressure: 28 mmHg
Field Volatility: mass loss of 11 – 26% of applied 1,3-D

Residues

Maximum contaminate Level
MCL: 0.5 ppb

No residues of concern in crops grown in soil fumigated with 1,3-D

No tolerances required

Toxicity

Acute oral LD_{50} in rats: 30 – 175 mg/kg
Acute dermal LD_{50} in rates: 1000 mg/kg
Acute dermal LD_{50} in rabbits:
333 – 540 mg/kg
Acute inhalation LC_{50} in rats: 900 ppm

Skin Irritant
Eye Irritant
Moderate skin sensitizer
Not teratogenic
Does not affect reproduction
Only relevant route of exposure inhalation

Ecotoxicity

Moderately toxic to fish and birds
Exposure potential is very unlikely

Product Safety

EPA IRIS Reference Concentration (RfC):
20 µg/m^3
Q* $(mg/kg/day)^{-1}$: 5.33×10^{-2}
Hypothetical cancer risk for occupational and residential populations:
1×10^{-5} to 1×10^{-8}

Conclusions

Active ingredient toxicity:
No dietary exposure and risks can be mitigated
Occupation and Residential Exposure:
Within range generally considered acceptable
Environmental Impact:
Minimal under normal use conditions
Environmental Fate:
Rapid degradation by several routes

Table of Contents

1 Identity of the Active Ingredient

1.1 Chemical Name

1,3-Dichloropropene (1,3-D)

1.2 Common Name

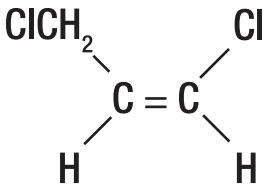
No official ANSI, BSI or ISO name Synonyms:
3-CHLOROALLYL CHLORIDE:
alpha-CHLOROALLYL CHLORIDE:
gamma-CHLOROALLYL CHLORIDE:
3-CHLOROPROPENYL CHLORIDE:
DCP:
DICHLOROPROPENE:
1,3-DICHLOROPROPENE-1:
1,3-DICHLOROPROPENE:
1,3-DICHLORO-3-PROPENE:
Dichloropropene, 1,3:
1,3-DICHLOROPROPYLENE:
NCI-C03985:
PROPENE, 1,3-DICHLORO-:
RCRA WASTE NUMBER U084:
TELONE® II

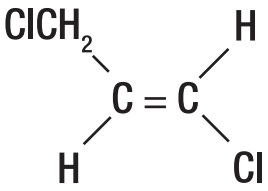
1.3 Molecular Weight

110.98 g/mole

1.4 Empirical and Structural Formula

Empirical formula: $C_3H_4Cl_2$
Structural formula:





1.5 CAS Registry Number

1,3-D: 542-75-6
Cis-Isomer: 010061-01-5
Trans-Isomer: 010061-02-6

2 Use

2.1 Purpose

Telone® soil fumigants are currently registered as a pre-plant soil treatment used to protect more than 120 vegetable crops, field crops, and nursery crops as well as planting sites for citrus trees, deciduous fruit trees, nut trees, and berry bushes and vines. Soil fumigation with Telone soil fumigants involves injecting liquid fumigant into the ground where it converts to a gas that moves primarily through the soil air space and dissolves into the film of water that surrounds soil particles. 1,3-dichloropropene (1,3-D), the active ingredient in Telone soil fumigants, controls certain soil-borne pests such as nematodes, viruses, bacteria and fungi that are present in the soil treatment zone at the time of application. 1,3-D will not control pests that are introduced into the soil after fumigation from sources such as contaminated soil, equipment, irrigation water and planting material.

2.2 General Information on Biological Spectrum of Activity

Telone soil fumigants may be applied as a pre-plant soil treatment to control the following types of plant parasitic nematodes: burrowing, citrus, cyst (sugar beet, soybean, carrot and wheat), dagger, lance, reniform, ring, root-knot, root lesion (meadow), spiral, sting, and stubby root. Telone soil fumigants can also be used to control garden centipedes (symphylans) and wireworms. Telone soil fumigants can suppress sugar beet *Rhizomania* disease, Granville wilt of tobacco, *Fusarium* wilt of cotton, *Verticillium* wilt of mint and potatoes, and aid in the control of bacterial canker of peaches. While 1,3-D and 1,3-D/chloropicrin combinations can be used to manage nematodes and soil borne diseases, weed control with these products is limited.

Soil fumigation with 1,3-D is a component in integrated pest management (IPM) programs. These IPM programs can include combinations with both chemical and nonchemical elements. Chemical combinations can include other fumigants such as chloropicrin and metam products and nonfumigants such as contact nematicides. Nonchemical components include crop rotation and resistant varieties.

3 Physical and Chemical Properties

3.1	**State**	Liquid
3.2	**Color**	Colorless to straw colored
3.3	**Odor**	Sweet penetrating
3.4	**Melting Point**	The melting point is lower than -50ºC.
3.5	**Boiling Point**	The boiling point of the *cis*-isomer is 104.1ºC. The boiling point of the *trans*-isomer is 112.6ºC.
3.6	**Vapor Pressure**	28 mm Hg at 20ºC.
3.7	**Density**	1.205 to 1.219 @ 20ºC. 1.200 to 1.214 @ 25ºC.
3.8	**Solubility**	
	a) In Water	The solubility of the *cis*-isomer in water is 2.18 g/L. The solubility of the *trans*-isomer in water is 2.32 g/L
	b) In Organic Solvents .	Probably soluble in most organic solvents
3.9	**Partition Coefficients**	
3.91	**K_{ow}**	The partition coefficients between n-octanol and water at 250ºC are: *Cis*-isomer - Log10K_{ow} = 2.09 at 0.0009M (K_{ow} = 122) *Cis*-isomer - Log10K_{ow} = 2.04 at 0.009M (K_{ow} = 110) *Trans*-isomer - Log10K_{ow} = 2.04 at 0.0009M (K_{ow} = 111) *Trans*-isomer - Log10K_{ow} = 2.01 at 0.009M (K_{ow} = 103)
3.92	**Henry's Law constant (water/vapor)**	*Cis*-isomer -1.8 X 10^{-3} m^3 atm/gmol (25ºC) *Trans*-isomer -1.05 X 10^{-3} m^3 atm/gmol (25ºC)

3.10 Stability

a) In Water

The hydrolysis half-lives of both *cis*- and *trans*- isomers determined in buffered solutions are identical over the pH range of 5 to 9 and are:

Half-Life (Days)			
pH	**10°C**	**20°C**	**30°C**
5	**51**	**11.3**	**3.1**
7	**51**	**11.3**	**3.1**
9	**51**	**11.3**	**3.1**

b) In Air

The air photolysis half-life for *cis*-1,3-D is 12 hours and 7 hours for *trans*-1,3-D.

Based upon daytime OH- radical concentration of about 2×10^6 cm^{-3} (24 hr average of 1×10^6 cm^{-3}).

c) In Organic Solvents

Not Applicable

4 Fate and Behavior in the Environment

4.1 Degradation and Mobility in Soil

Aerobic and Anaerobic Soil Metabolism (Half-Life)

Degradation half-life in soil is rapid to moderately rapid at about 1 week in loamy and clayey soils, and less than 3 weeks in sandy soils, in aerobic and anaerobic soil conditions (with and without free oxygen).

Aerobic Degradation Products and Degradation Rates

3-Chloroallyl alcohol in field dissipation studies declined from a maximum of 410 ppb in the 0.66 to 0.81 meter layer at 7 days post-treatment to <10 ppb in any soil layer at 71 days. In aerobic metabolism studies, metabolites identified have been 3-chloroallyl alcohol; 3-chloracrylic acid; numerous carboxylic acids, such as acetic acid, adipic acid, butyric acid, chloroacetic acid, 4-chlorobutyric acid, fumaric acid, glycolic acid, hexanoic acid, lactic acid, malic acid, malonic acid, 2-methylmalonic acid, oxalic acid, propionic acid and succinic acid. Soil degradation is characterized as rapid with a half-life of 6-30 days in clay to sand soils and 1-2 days in clay soils.

1,3-D aerobic soil metabolism (162-1)

Type Half life	(days)	%OM	ºC	pH	Comments
Spier SL	22/ — (*cis/trans*)	11.6	15		van Dijk (1974)
Spier SL	37/ —	11.1	15		van Dijk (1974)
Haren SL	22/26	3.6	15		van Dijk ((1974)
Haren SL	22/26	3.6	15	5.0	van Dijk ((1974)
Bogercie SL	20/20	14.8	20	5.6	van Dijk ((1974)
Bant	23/24	5.5	20	3.9	van Dijk (1974)
Wieringerwerf	3/3	1.1	20	6.8	van Dijk (1974)
Baexem	3.5/3	1.7	20	7.2	van Dijk (1974)
Wieringerwerf	7.5/7.5	2.6	20	7.6	van Dijk (1974)
Warffum	8/8	1.8	20	7.2	van Dijk (1974)
Warffum	5/7	1.8	20	7.1	van Dijk (1974)
Catlin SiL	12	3.6	20	6.6	GH-C 2967
Fuquay LS	54	1	25	4.7	GH-C 29675
Wahiawa SCL	2	3.9	25	4.7	Wolt et al, 1992
Sandy soils	Mean *(cis)* =19; Range =11-25	7.3; 1.9-14.8	20	4.6; 3.9-5.3	van Dijk (1980)
	Mean *(trans)*=15; Range = 5-23				
Clay soils	Mean *(cis)*=6.5; Range = 3-11	1.8; 1.1-2.6	20	7.3; 6.8-7.7	van Dijk (1980)
	Mean *(trans)*=5.5 Range = 3-8				

SL=Sandy Loam SiL=Silty Loam SCL=Sandy Clay Loam LS=Loamy Sand

Anaerobic Degradation Products and Degradation Rates

Under anaerobic conditions, 1,3-D had an affinity for the water phase over the organic phase. Half-life range for 1,3-D was 2.4 days at 25ºC, and 7.7 days to 9.1 days at 15ºC. 3-Chloroallyl alcohol and propionic acid were above 10% of applied parent.

1,3-D anaerobic soil metabolism study (162-2)[1]					
Type	Half life (days)	%OM	ºC	pH	Comments
Catlin SiL	9.1	3.3	15	5.9	GH-C 1861
Catlin SiL	2.4	3.3	25	5.9	GH-C 1861
Cecil SL	7.7	1.6	15	5.7	GH-C 1861
Cecil SL	2.4	1.6	25	5.7	GH-C 1861

[1]Degradates found: 3-chloroallyl alcohol and propionic acid.

SiL = Silty Loam SL = Sandy Loam

Soil Partition Coefficient (K_d)

K_d is an indication of the affinity of the compound for soil, and indicates the relative mobility of a compound in soil (0.1 to 10 is potentially very mobile). The K_d for 1,3-D in loamy sand was 0.23 (Fuquay 1s); sand was 0.32 (Myakka fs); clay was 0.42 (Texas soil) and 1.09 (Hawaii soil).

K_{oc} is the K_d divided by the organic carbon content of the soil (0.1 to 100 indicates a potentially very mobile compound). Average K_{oc} values for 1,3-D were 20 for sand, 25 for loamy sand, and 41 and 42 for two clay soils.

1,3-D soil sorption coefficients (163-1)[1]					
Type	K_d	K_{oc}	%OC	pH	Comments
Fresh Column Leaching					
Fuquay LS	0.23	25	0.92	4.7	GH-C 2018
Myakka FS	0.32	20	1.64	5.4	GH-C 2018
Hudalgo C	0.42	43	1.00	8.4	GH-C 2018
Wahiawa C	1.09	41	2.68	5.0	GH-C 2018
Aged Column Leaching					
Fuquay LS	2.25	352	0.64	4.7	GH-C 2879

[1]Data derived from column leaching methods.

C=Clay LS=Loamy Sand FS=Fuquay Sand

1,3-D Hydrolysis (161-1)			
°C	pH	Half life (days)	Comments
10	5,7,9	51	McCall (1987)
20	5,7,9	11	McCall (1987)
30	5,7,9	3	McCall (1987)

Soil Rf Factors – The Rf factor is a retention factor estimated in laboratory leaching studies. In 30 cm long columns of sand, loamy sand, and Texas clay soils, leached with >25˝ water, 1.9-4.6% applied radioactivity remained in soils and 79-84% was in leachate. Due to short study duration, no metabolites were detected in leached column studies.

Terrestrial Field Dissipation – In a field plot of sandy soil in California, 1,3-D, applied at 342 lb ai/A, declined from a maximum of 130,000 ppb in the 0.30 m to 0.45 m layer immediately after treatment, to <10 ppb (detection limit) in any soil layer at 71 days. Half-life was 0.6 to 5.6 days. Maximum depth of detectable residues was 9 ft mainly due to diffusion rather than leaching.

A northern Florida field study applied 345 lbs of 1,3-D per acre to a loamy sand soil. A two-phase degradation curve was observed; half-life was 3 day in the first two weeks, then a 81- to 84-day half life was calculated through day 562 of the study. Maximum depth of detectable residues was 10 ft, due to diffusion rather than leaching. 3-Chloroallyl alcohol metabolite followed patterns of movement similar to 1,3-D and dissipated even more rapidly than parent 1,3-D.

4.2 Behavior in Water

Maximum Contaminant level (MCL) for 1,3-D is 0.5 ppb

Solubility – 1,3-D is moderately soluble in water.

1,3-D Hydrolysis rate –1,3-D has been shown to hydrolyze in aqueous solution at a rate dependent upon water temperature. At 20ºC (an expected annual average water temperature in southern Florida) the half-life was 11 days. Hydrolysis rate will proceed at a faster rate at higher temperatures and slower at lower temperatures. 3-Choroallyl alcohol is an hydrolysis product; it also is expected to degrade rapidly.

4.3 Behavior in Air

1,3-D has a high vapor pressure and is a volatile compound.

Air photolysis – 1,3-D reacts with sunlight only in the presence of free OH- radicals. A study of half-life with a GE sunlamp was 0.5 to 3.3 days. Estimated half-life, based upon expected daytime levels of OH- radicals, is 7 to 12 hours.

Field Volatility – A study of a clay loam soil in Imperial Valley of California showed about 11% of applied 1,3-D volatilized in an 8-day period. In a recent study in Salinas Valley of California, about 25% of applied 1,3-D volatilized from a sandy loam soil. 1,3-D flux (volatility) rates vary as a function of soil moisture conditions, soil temperature conditions, depth of injection, quality of soil sealing, application rates, time-of-day and number of days after application. Peak emissions occurred during nighttime periods. Maximum flux rates were detected two to five days following fumigation. The soil was not tarped in these studies.

4.4 Modes of Dissipation for 1,3-D

- Gaseous diffusion throughout the soil and flux through soil/air interface
- Hydrolysis in water, including soil pore water
- Biological metabolism by aerobic and anaerobic microorganisms

5 Residues In Food

5.1 Plant Residues and Metabolism

Studies with radiolabeled *cis*- and *trans*- 1,3-D have shown that the compounds are rapidly degraded and incorporated into natural plant products. These products include basic plant components such as fatty acids, proteins, pigments, organic acids, simple sugars and carbohydrates, and plant structural components (cellulose and lignin). These studies have also demonstrated that no detectable residues of 1,3-D are present in wheat and soybean forage, radish and carrot foliage or roots, lettuce, tomato fruit, wheat straw or grain, or soybean beans. Therefore, no residues of concern are identifiable in the crops grown in soil fumigated with 1,3-D.

Field residue trials have confirmed the absence of residues of 1,3-D in carrots, onions, grapes, cantaloupe, broccoli, lettuce, tomatoes, potatoes, pineapples, sugar beets, soybeans, oranges, peaches, cottonseed, and peanuts at a limit of quantitation of 0.01 µg/g.

5.2 Domestic Animal Residues and Metabolism

A metabolism study with radiolabeled *cis*- and *trans*- 1,3-D in lactating goats demonstrated that 1,3-D and/or its metabolic products were rapidly excreted or expired following multiple dosing at 1300 times the potential dietary exposure level for five days. Approximately 4% of the total dose was secreted in the milk and a minor portion (approximately 1% of the dose) remained associated with the body tissues 24 hours after the last dose. The liver was shown to contain the highest residue level, representing approximately 0.7% of the total dose, at a tissue concentration of about 0.9 mg 1,3-D equivalents/kg. The residues from the tissues and milk contained no parent compound and were composed of animal natural products such as glucose, glycogen, protein, fatty acids, glycerol, lactose, and amino acids.

A poultry metabolism study with radiolabeled 1,3-D demonstrated that 1,3-D and/or its metabolic products were rapidly excreted or expired following multiple doses at 3500 times the potential dietary exposure level for seven days. A minor portion (less than 0.3%) of the total dose was found in the eggs and only about 3.3% of the total dose remained associated with the body tissues 24 hours after the last dose. The liver was shown to contain the highest residue level, representing approximately 0.3% of the total dose, at a tissue concentration of about 0.6 mg 1,3-D equivalents/kg. This residue contained no parent compound and was composed of at least one metabolite.

Muscle samples contained the highest percentage (0.8%), of the total dose found in the chicken tissues, representing a concentration of about 0.2 mg 1,3-D equivalents/kg. Muscle residue contained no parent compound and was composed of at least two metabolites. The residue from fat contained about 0.14 mg/kg parent and at least one metabolite. Eggs contained residues of parent (0.01 mg/kg), two metabolites and components of animal natural products such as protein, fatty acids and amino acids. Considering the low residue levels found in tissues and eggs, as well as the greatly exaggerated potential dietary exposure rate used for dosing in this metabolism study, the identified residues were determined to be of no toxicological concern. These studies show that 1,3-D and/or its metabolites are not likely to accumulate in animal tissues, eggs, nor be secreted in milk to a significant extent following oral ingestion of crops grown on soil fumigated with 1,3-D. Therefore, no meat, egg or milk tolerances are required of 1,3-D.

6 Mammalian Metabolism

6.1 Metabolism

A series of studies have been conducted to determine the pharmacokinetic behavior and metabolism of 1,3-D in rats, mice and humans. Data indicate that 1,3-D is absorbed from the skin, respiratory tract and gastrointestinal tract. Following absorption, both the *cis*- and *trans*- isomers of 1,3-D are rapidly eliminated from the bloodstream of rats (half-life approximately 2–4 min) and humans (half life <10 min). A number of studies have determined the predominant routes of excretion of radioactivity in male and female rats following a single dose or repeated oral doses of *cis*-, *trans*- or racemic 1,3-D to be the urine (*cis*-, 82-84%; *trans*-, 56–61%; racemic, 51–65%), feces (*cis*- or *trans*-, 2–3%; racemic, 6–18%), and expiration as $C0_2$ (*cis*-, 2–5%; *trans*-, 23–24%; racemic, 15–25%). There were no remarkable sex-related differences in excretion routes or kinetics observed in rodents.

Excretion and distribution of 1,3-D was independent of dose in rats administered up to 50 mg/kg via gavage, with greater than 80% of the administered dosages being excreted within 24 hours of dosing. A similar pattern of excretion and distribution occurred in mice administered up to 100 mg/kg racemic compound. Humans were observed to excrete approximately 90% of an estimated dose of inhaled racemic 1,3-D within 36 hours of exposure. The primary urinary excretion product in rats and mice is the mercapturic acid conjugate of 1,3-D and its corresponding sulfoxide. Mice also excrete a significant portion of absorbed 1,3-D as a mercaptoacetic acid conjugate. The mercapturic acid conjugate has been identified in the urine of human subjects exposed to mixed isomer vapors of 1,3-D under field application or laboratory conditions. Humans have been estimated to excrete approximately 45% and 14% of absorbed c*is*- and *trans*- isomers of 1,3-D, respectively, as their respective mercapturic acid metabolites.

7 Toxicity of the Technical Active Ingredient

7.1 Acute Oral Toxicity

Rat LD_{50}:

Male (Fischer 344) . . . . . 300 mg/kg
Female (Fischer 344) . . . . . 224 mg/kg
Male (Sprague-Dawley) . . . . . 130 mg/kg
Female (Sprague-Dawley) . . . . . 110–250 mg/kg
Male (Wistar) . . . . . 560 mg/kg
Female (Wistar) . . . . . 510 mg/kg

Mouse LD_{50}

Undefined Sex (CD-1) . . . . . 215 mg/kg
Male (JCL:ICR) . . . . . 640 mg/kg
Female (JCL:ICR) . . . . . 640 mg/kg

Acute Oral Toxicity in Human (Accidental)

Two fatalities have been reported involving 1,3-D following the accidental ingestion of formulated products. Victims' symptomology included: abdominal pain and vomiting, adult respiratory distress syndrome, hematological changes, hepatorenal impairment, coma, and muscular twitching.

7.2 Acute Dermal Toxicity

Rabbit (New Zealand White) LD_{50}:

Male and Female . . . . . 504 mg/kg
Male . . . . . 333 mg/kg
Female . . . . . 333 mg/kg

Rat (Wistar) LD_{50}:

Male . . . . . .1000 -> 1211
Female . . . . . 1300–2000 mg/kg

Mouse (JCI:ICR) LD_{50}:

Male . . . . . >1211 mg/kg
Female . . . . . >1211 mg/kg

7.3 Acute Inhalation Toxicity

Rat LC_{50} (4-hour):

Male (Fischer 344) . . . . . .904 ppm
Female (Fischer 344) . . . . . .855–1035 ppm
Male and female (Wistar) . . . . . .729 ppm

Acute (Accidental) Inhalation Toxicity in Humans:

Forty-six people were treated for exposure to 1,3-D fumes following a traffic accident in 1975 involving spillage of 4500 liters of a formulated product. Twenty-four of these, three of which had lost consciousness, were hospitalized overnight with symptoms including; headache, irritation of mucous membranes and chest discomfort. All patients took showers and were given intravenous fluids and three received oxygen and corticosteroids because of chest pain and cough. Eleven of 41 persons tested had slightly higher than average serum SGOT and/or SGPT values which reverted to normal within 48-72 hours, except for five which still had slightly higher than average SGOT values. Follow-up interviews with patients 1-2 weeks later revealed symptoms including headache, abdominal and chest discomfort and malaise. One was diagnosed as having had pneumonia. Symptoms were reported more frequently in those most heavily exposed to the fumes. Patient interviews conducted approximately two years after the accident revealed complaints of headache, chest pain or discomfort and "personality changes" (fatigue, irritability, difficulty in concentrating or decreased libido). Two had undergone cardiac catheterizations but their arteriograms were normal. There was no correlation of these claims of long-persisting symptoms with intensity of exposure.

7.4 Skin Irritation

Rabbit (New Zealand White):

24-Hour Occluded . . . . . Severe
4-Hour Occluded . . . . . Moderate

Human (Accidental):

Accidental contact of 1,3-D with skin, especially if occluded, reportedly may cause edema, redness, and, if prolonged, necrosis of the skin.

7.5 Eye Irritation

Rabbit (New Zealand White):

Slight to Moderate; Reversible

7.6 Skin Sensitization

Guinea pig (modified Buehler):

Moderate Positive

Human (Accidental):

There has been one documented case in which 1,3-D was believed to have caused a contact hypersensitivity in a repeatedly exposed farmer.

7.7 Sub-chronic Toxicity

Rat (90-day oral):

Fischer 344 rats were administered up to 100 mg/kg/day of a stabilized preparation (microencapsulated) of racemic 1,3-D via their diets. Direct, treatment-related effects observed

included; decreased body weight and feed consumption at ⩾15 mg/kg/day dosage in males and ⩾50 mg/kg/day dosage in females. The nonglandular portion of the stomach was identified as a target organ. The no-observed-effect level (NOEL) was 5 mg/kg/day in both sexes.

Rat (90-day inhalation):
Fischer 344 rats were exposed to up to 150 ppm racemic 1,3-D vapor for 6 hr/day, 5 days/wk. Treatment-related decreases in body weights were observed in both sexes at 90 ppm vapor exposure. The respiratory and olfactory epithelia of the nasal mucosa were identified as target tissues. The NOEL was 10 ppm vapor. In a subsequent study, Fischer 344 rats were exposed to up to 90 ppm *cis*- 1,3-D vapor for 6 hr/day, 5 days/wk. Treatment-related decreases in body weights were observed in both sexes of high exposure group animals. The respiratory and olfactory epithelia of the nasal mucosa were identified as target tissues. The NOEL was 30 ppm vapor.

Mice (90-day oral):
B6C3F1 mice were administered up to 175 mg/kg/day of a stabilized preparation (microencapsulated) of racemic 1,3-D via their diets. Direct, treatment-related effects observed included; decreased body weight at ⩾15 mg/kg/day dosages in both sexes. Histologically there was evidence of decreased liver glycogen at ⩾15 mg/kg/day dosages in males and decreased lipid content of kidney tubular cells in high dose group males. The NOEL was 15 mg/kg/day in both sexes.

Mouse (90-day inhalation):
B6C3F1 mice were exposed to up to 150 ppm racemic 1,3-D vapor for 6 hr/day, 5 days/wk. Treatment-related decreases in body weights were observed in both sexes of high exposure group animals. The epithelia of the nasal mucosa of both sexes and the transitional epithelium of the urinary bladder of females were identified as target tissues. The NOEL was 10 ppm vapor.

Human (occupational exposure):
There have been several claims of slight increases in the excretion of hepatic and/or renal enzymes in urine of soil fumigant applicators over a working day or growing season. Subsequent studies have failed to demonstrate any change in clinical chemistry or urinary parameters in humans suggestive of an untoward effect of exposure.

7.8 Chronic Toxicity

Rat (two-year oral gavage):
In a chronic toxicity and oncogenicity study, Fischer 344 rats were administered dosages of 25 or 50 mg/kg/day of an epichlorohydrin-stabilized formulation of racemic 1,3-D via gavage 3 days/week. A treatment-related increase in malignant tumors of the nonglandular portion of the stomach and benign liver tumors was observed in one or both dose groups of males. Malignant forestomach tumors were observed in high dose group females. Hyperplasia of the epithelium of the stomach mucosa accompanied tumor formation in this tissue. No NOEL was established.

Rat (two-year oral dietary):
In a chronic toxicity and oncogenicity study, male and female Fischer 344 rats were administered dosages of 2.5, 12.5 or 25 mg/kg/day of an epoxidized soybean oil (ESO)-stabilized formulation of racemic 1,3-D as a microencapsulated formulation via animal feed for up to two years. A statistically-identified increase in benign liver tumors was identified in high dose group males and females. Numbers of benign liver tumors were also increased relative to the historical control incidences of this tumor type in males ingesting 12.5 mg/kg/day 1,3-D. No other tumorigenic response was observed. The only clear treatment-related non-neoplastic changes noted in treated rats was a significant depression in body weights and a slight hyperplasia of the nonglandular stomach mucosa in both sexes of rats ingesting 12.5 or 25 mg/kg/day 1,3-D. An increased incidence of normally occurring eosinophilic foci of cells was also noted in the livers of all treated groups of rats, however, this change was equivocal in terms of its toxicological significance. The NOEL was 2.5 mg/kg/day in both sexes.

Rat (two-year inhalation):
In a chronic toxicity and oncogenicity study, Fischer 344 rats were exposed to up to 60 ppm of an ESO-stabilized formulation of racemic 1,3-D for six hrs/day, five days/week, for up to two years. No tumorigenic response was observed in either sex of exposed rats. A treatment-related degeneration of the nasal mucosal epithelium was observed in high exposure group males and females. The NOEL was 20 ppm vapor in both sexes.

Mouse (female only; two-year oral gavage):
In a chronic toxicity and oncogenicity study, B6C3F1 mice

were administered dosages of 50 or 100 mg/kg/day of an epichlorohydrin-stabilized formulation of racemic 1,3-D via gavage 3 days/week. A treatment-related increase in malignant and/or benign tumors of the nonglandular portion of the stomach, urinary bladder, and lungs was observed in one or both dose groups of females. Hyperplasia of the epithelium of the stomach and urinary bladder mucosa accompanied tumor formation in these tissues. No NOEL was established.

Mouse (two-year oral dietary):
In a chronic toxicity and oncogenicity study, male and female B6C3F1 mice were administered dosages of 2.5, 25 or 50 mg/kg/day of an ESO-stabilized formulation of racemic 1,3-D as a microencapsulated formulation via animal feed for up to two years. A treatment-related decrease in body weights was noted in male and female mice ingesting 25 or 50 mg/kg/day 1,3-D. No other treatment-related effects, including tumors, were observed in treated mice. The NOEL was 2.5 mg/kg/day in both sexes of mice.

Mouse (two-year inhalation):
In a chronic toxicity and oncogenicity study, B6C3F1 mice were exposed to up to 60 ppm of an ESO-stabilized formulation of racemic 1,3-D six hrs/day, five days/wk, for up to two years. The only tumorigenic response observed was an increased incidence of benign tumors in the lungs of high exposure group males. Nontumorigenic effects included hyperplasia of the epithelium of the nasal and/or urinary bladder mucosa in males and females at (20 ppm vapor and hyperplasia of stomach mucosa in high exposure group males. Changes also were observed in the liver and kidneys of high dose group males indicative of glycogen and lipid depletion, respectively. The NOEL was 5 ppm vapor.

Mouse (18-month dermal):
In a chronic skin painting study, female Ha:ICR Swiss strain mice were exposed to 122 mg (>3000 mg/kg/day) *cis*-1,3-D (stabilizer unknown), three times/wk, for 18 months. No increased incidence of tumor, localized or distal tissues, was reported.

Mouse (initiation-promotion):
Cis-1,3-D (stabilizer unknown) was not observed to be an initiator in a skin painting initiation-promotion assay conducted using female Ha:ICR Swiss strain mice with a phorbol ester promoter.

Dog (12 month oral):
Male and female Beagle dogs were administered up to 15 mg/kg/day of a stabilized preparation (microencapsulated) of racemic 1,3-D via their diets. The body weights of high dose level males were decreased relative to controls. A treatment-related hypochromic, microcytic anemia, without bone marrow depletion, occurred in both sexes of dogs ingesting 15 mg/kg/day 1,3-D and a slight inflammation of the tongue was also observed in several high dose level males. The NOEL was 2.5 mg/kg/day in both sexes.

7.9 Reproductive Toxicity

Rat Teratology:
1,3-D (racemic) was not embryotoxic or teratogenic in bred Fischer 344 rats exposed to up to 120 ppm vapors, six hours/day, during gestation days 6–15.

Rabbit Teratology:
1,3-D (racemic) was not embryotoxic or teratogenic in inseminated New Zealand White rabbits exposed to up to 120 ppm vapors, six hours/day, during gestation days 6–18.

Rat Multigenerational Reproduction:
Exposure of male and female Fischer 344 rats to up to 90 ppm racemic 1,3-D vapors for two generations did not adversely affect reproduction or neonatal growth or survival of pups.

Mouse Sperm Morphology:
No treatment-related changes in testes weight, sperm count or sperm morphology occurred in mice 30 days after being injected (ip) with up to 600 mg/kg/day 1,3-D for 5 days.

Human Reproductive Evaluation:
A fertility study of 64 employees engaged in the production of chlorinated 3-carbon compounds, including 1,3-D, revealed no effects upon hormone levels (LH, FSH, testosterone), sperm count, sperm motility and % normal and abnormal sperm regardless of duration or magnitude of exposure.

7.10 Genotoxicity

Bacterial Assays:
Early studies reporting mutagenic responses for 1,3-D in *S. typhimurium* and *Saccharomyces* tester strains were confounded by the presence of a mutagenic stabilizing agent, epichlorohydrin, in the formulations used for the generation of mutagenic oxidation products of 1,3-D during

gas chromatographic purification operations. Subsequent studies using appropriately purified 1,3-D were weakly positive only in the presence of liver microsomes. Addition of cytosolic enzymes, presumably containing glutathione transferase, abolished activity.

Mammalian Cell Assays:
Negative results have been obtained for a recent ESO-stabilized racemic formulation of 1,3-D in the Chinese Hamster Ovary HGPRT forward mutation and rat hepatocyte unscheduled DNA synthesis assays. Gas chromatographically purified *cis*- and *trans*- 1,3-D isomers induced unscheduled DNA synthesis in HeLa cells. 1,3-D (stabilizer unknown) reportedly caused DNA fragmentation and repair in V79, rat and human hepatocytes in vitro. 1,3-D (stabilizer unknown) also reportedly caused chromosomal structural aberrations in cultured CHL and CHO cells.

Drosophila Assays:
When fed to Drosophila at a concentration of 5750 ppm, an epoxide-stabilized 1,3-D formulation caused an increased incidence of sex-linked recessive lethal mutations but not reciprocal translocations.

In Vivo Mammalian Assays:
Negative results were obtained in a mouse bone marrow micronucleus test at a dosage of up to 380 mg/kg ESO-stabilized 1,3-D formulation. Negative results also have been obtained for a 1,3-D formulation (stabilizer unknown) in a host-mediated bacterial mutagenicity assay in mice. 1,3-D (stabilizer unknown) was reported to be positive for somatic cell mutations in a mouse spot test at a dosage of 150 mg/kg (ip) and to cause single strand breaks in DNA of several tissues and DNA repair in hepatocytes in rats dosed with up to 125 mg/kg (ip or po).

7.11 Neurotoxicity

The molecular structure of 1,3-D does not suggest that any neurotoxicity is likely to occur as a result of exposure. There is no evidence from the battery of toxicity studies with 1,3-D at sublethal dosages to suggest that it is likely to cause a neurotoxic effect.

8 Risk and Safety Information on the Products

8.1 Occupational Exposure and Risk

A comprehensive worker exposure evaluation was conducted in three major U.S. Telone® soil fumigant use areas to evaluate potential 1,3-D exposure encountered by workers loading, and/or applying Telone® II and Telone® C-17 soil fumigants or entering fields recently treated with Telone II. The intent of the study was to allow for an analysis of the effectiveness of different engineering controls and personal protective equipment (e.g. respiratory protection) in managing exposures encountered by workers handling Telone soil fumigants and to quantify the exposure encountered by Telone product handlers.

The average measured 1,3-D air concentrations (derived from either air monitoring or biological monitoring) that are representative of the selected, typical exposure scenarios compared to the OSHA PEL (8 hour time-weighted average of 1 ppm) and the IRIS Reference Concentration (RfC, 20 $\mu g/m^3$) indicate that Telone product loaders, applicators and workers re-entering treated fields are likely to be without an appreciable risk of health effects during a lifetime. Further, the calculated hypothetical, cancer risks of 1.0×10^{-6} to 4.8×10^{-6} for product loaders and 4.7×10^{-6} to 3.5×10^{-5} for product applicators and 2.1×10^{-6} to 1.5×10^{-5} for re-entry workers are all within the range of cancer risks (10^{-4} to 10^{-6}) considered acceptable for worker populations.

The results of the risk assessment indicate the use of dry disconnects for product loaders and spill control for product applicators are practical, useful exposure mitigation measures that can be employed on a commercial basis and help provide a safe environment for workers.

8.2 Residential Exposure and Risk

Off-site air concentrations of 1,3-D collected in Washington, North Carolina and Arizona were in simplistic Monte Carlo analyses to estimate high end exposure (HEE) and risk for populations living near fields treated with Telone soil fumigant. Distributions of 1,3-D concentration, breathing rate, personal/outdoor activity and residence time were used as input for the exposure simulations. Estimates of lifetime average daily dose (LADD) and annual average daily dose (AADD) were calculated for each region. An exposure range of interest (defined as the 50th to 95th percentile estimates of LADD and AADD) was used to estimate the range of risks for each site.

Exposure estimates for Washington were 0.0123–0.0421 µg/kg/day (LADD) and 0.0573–0.1924 µg/kg/day (AADD). For North Carolina, analogous ranges are 0.0013–0.0058 µg/kg/day (LADD) and 0.0040–0.0176 µg/kg/day (AADD). For Arizona, the ranges are 0.0071–0.0594 µg/kg/day (LADD) and 0.0219–0.1890 µg/kg/day (AADD).

The potential for health effects (e.g. tumorigenicity) to arise through a threshold mechanism of action was judged in this assessment by comparing AADD estimates to the IRIS reference concentration for potential 1,3-D of 5.7 µg/kg/day (equivalent to 20 $\mu g/m^3$). Based on this comparison, hypothetical populations living near a field in Washington were estimated to encounter exposures representing 1 to 3% of the RfC. Similar populations living near a field in North Carolina were estimated to encounter exposures representing 0.1 to 0.3% of the RfC and those in Arizona were estimated to experience 0.4 to 3% of the RfC. Thus, in each region, 95% of the HEE population is predicted to experience exposures that represent less than 3% of the RfC, a chronic inhalation guidance level developed by the EPA to protect the general population (including sensitive subgroups).

The potential for health effects (e.g. tumorigenicity) to arise through a non-threshold mechanism of action also was evaluated in this assessment. Theoretical lifetime individual cancer risks were calculated for each area of interest using the Monte Carlo estimates of LADD. Based on this calculation, hypothetical populations living near a field in Washington were estimated to experience risks ranging from 6×10^{-7} to 4×10^{-6}. Estimated risks for similar populations living near a field in North Carolina were on the order of 6×10^{-8} to 6×10^{-7} and those for populations living near a field in Arizona ranged from 3×10^{-7} to 6×10^{-6}. Thus, 95% of the HEE population in each region is predicted to experience less than 10^{-5} Risk, a level well within the range (10^{-4} to 10^{-6}) generally considered acceptable.

Based on the information contained in this assessment, normal agricultural use of Telone soil fumigants is unlikely to result in a significant risk of toxicity or carcinogenicity to human populations living in the vicinity of treated fields.

Telone soil fumigants are moderately toxic to birds and fish. To avoid injury to fish and other wildlife, do not spill or empty

9 Wildlife

Telone soil fumigants into streams, ponds, or other bodies of water. Do not load Telone soil fumigants near a body of water.

9.1 Birds

Avian exposure to 1,3-D would be very unlikely when used according to the label.

Mallard duck . . . . . LD_{50} (8 day) . . . . . >10,000 mg/kg diet
Bobwhite . . . . . LD_{50} (8 day) . . . . . >10,000 mg/kg diet

9.2 Fish

Aquatic organism exposure to 1,3-D would be very unlikely when used according to the label.

Bluegill . . . . . LD_{50} (96 hrs) . . . . . .7.1 mg/L
Rainbow trout . . . . LD_{50} (96 hrs) . . . . . .3.9 mg/L
Mysid . . . . . MATC (28 days) . . . . . >4.09 mg/L

9.3 Other Aquatic Organisms

Daphnia . . . . . LD_{50} (48 hrs) . . . . . .6.2 mg/L

9.4 Bees

1,3-D is not toxic to bees when used as directed.

10 Publications

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Notes

Dow AgroSciences

Telone is a Restricted Use Pesticide.

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