1,3-Dichloropropene

By Caroline Cox

It's April 1990 in California's Central Valley. Routine air monitoring for a common soil fumigant measures concentrations in the air that are almost 900 times the concentration that California's Department of Health Services has previously estimated to cause one cancer case in every hundred thousand exposed persons. The highest concentrations are measured at a junior high school in Merced where the chemical has recently been used on nearby sugar beet and sweet potato fields. The chemical is found in the air at five other sites within the county during the first two weeks of April.[1]

California officials immediately suspend use of the chemical,[2] but it is worth noting that the chemical has been used as a pesticide for almost twenty-five years[3] at rates as high as 1150 pounds per acre[2] and other episodes of high air concentrations may well have occurred. What's the chemical? 1,3-Dichloropropene (Figure 1), marketed under the trade names Telone and D-D 92.

History, Uses, and Manufacturing Data

1,3-Dichloropropene has been registered as a pesticide in the U.S. since 1966. It is manufactured by DowElanco for sale in the U.S. as Telone II and by Shell International Chemical Co., Ltd as D-D 92 for sale in other countries.[4] It is a general biocide, and is used primarily to kill nematodes but also plant diseases, insects, and weeds on potatoes, tomatoes, tobacco, pineapple, and other vegetable and orchard crops. (See Figure 2.)

Usage rates range from six[5] to 1150[2] pounds per acre. (See Figure 3.) In 1989, the U.S. Environmental Protection Agency (EPA) estimated that between 35 and 45 million pounds of 1,3-dichloropropene were used annually in the United States. By weight (based on 1989 data), it is the fifth most abundantly used pesticide in the U.S.[6]

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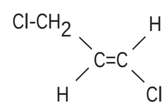
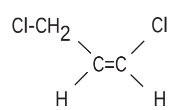
Chemistry and Mode of Action

1,3-Dichloropropene is a colorless, sweet smelling liquid that evaporates easily. There are two isomers (molecules composed of the same atoms, but structurally different) called the cis and trans isomers. The two isomers differ in the position of the atoms attached to the carbon-carbon double bond in the middle of the 1,3-dichloropropene molecule. (See Figure 1) and have similar, but not identical, chemistries. Different formulations contain different mixtures of the isomers. Telone II, for example, is a mixture of between 48 and 53 percent of the cis isomer and between 42 and 45 percent of the trans isomer.[7]

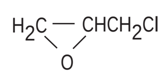
Figure 1
1,3-Dichloropropene and Some of its "Inert" Ingredients and Contaminants



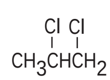
trans-1,3-Dichloropropene



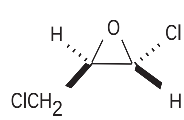
cis-1,3-Dichloropropene



Epichlorohydrin



1,2-Dichloropropane



An oxirane

1,3-Dichloropropene acts to kill nematodes in a manner similar to other volatile nematocides, including methyl bromide, ethylene dibromide, and chloropicrin. The 1,3-dichloropropene molecule reacts with an unidentified vital enzyme system (or systems) at a site on the enzyme containing sulfhydryl (sulfur + hydrogen), ammonia, or hydroxyl (oxygen + hydrogen) ions. A substitution reaction occurs in which the 1,3-dichloropropene molecule minus one of its chlorine atoms replaces one of the hydrogen atoms on the enzyme. As a result, the enzyme ceases to function properly. Hyperactivity of the nematode then occurs, followed by paralysis and death.[8]

Acute Toxicity

People exposed occupationally or from spills to 1,3-dichloropropene have suffered chest pains, coughing, breathing difficulties and skin rashes.[7,9] Irritation of the eyes and respiratory tract, liver and kidney damage, and cardiac arrhythmias are also symptoms of 1,3-dichloropropene exposure.[10]

In rats, single oral doses of Telone II caused lung damage. In addition, stomach ulcerations and bleeding of livers and intestines were found in rats after a single oral dose of cis-dichloropropene. Dermal exposure caused many of the same effects, but also caused inflammation and itching of the skin in rats, rabbits, and guinea pigs and incoordination, lethargy, and salivation in rats and rabbits.[7] If applied to rabbits' eyes, Telone II caused eye irritation, damage to the cornea, and temporary loss of vision.[3]

The most toxic route of exposure to 1,3-dichloropropene in laboratory animals appears to be oral, with LD_{50}s (the dose required to kill 50 percent of population of test animals) of the cis isomer in female rats as low as 117 milligrams per kilogram (mg/kg) of body weight. If the LD_{50}s for humans are similar, a little over a teaspoon would be a fatal dose. Because the chemical evaporates quickly, the most typical route of exposure is probably through inhalation. Concentrations required to kill 50 percent of test animals (LC_{50}) of close to 900 mg/kg have been recorded in rats breathing air contaminated with Telone II. For rabbits exposed dermally to Telone II, the LD_{50} is 333 mg/kg.[7]

Chronic Toxicity

Studies of applicators who fumigated Dutch flower bulb fields with 1,3-dichloropropene have measured changes in five parameters of liver and kidney function.[11] A California study of applicators found evidence of kidney damage in nine of the fifteen workers tested.[12]

In laboratory animals, long-term exposure to 1,3-dichloropropene has caused a variety of adverse effects. Rats inhaling Telone II suffered from swollen and congested lungs and damage to cells in the lining of the lungs. In mice and rats, inhalation of 1,3-dichloropropene caused depressed growth rates (up to 20 percent in rats and 12 percent in mice).[13] Telone administered to rats by gavage (through a feeding tube into the stomach) for 13 weeks caused an increase in kidney weights relative to body weights. The no observable effect level (NOEL; the highest dose showing no adverse effect) for this effect was 3 milligrams per kilogram (mg/kg) per day. This is the lowest NOEL observed in any of the tests required to register 1,3-dichloropropene with EPA. If humans are affected at a similar dose, daily exposure of less than 1/20 of a teaspoon would be sufficient to alter kidney weight.

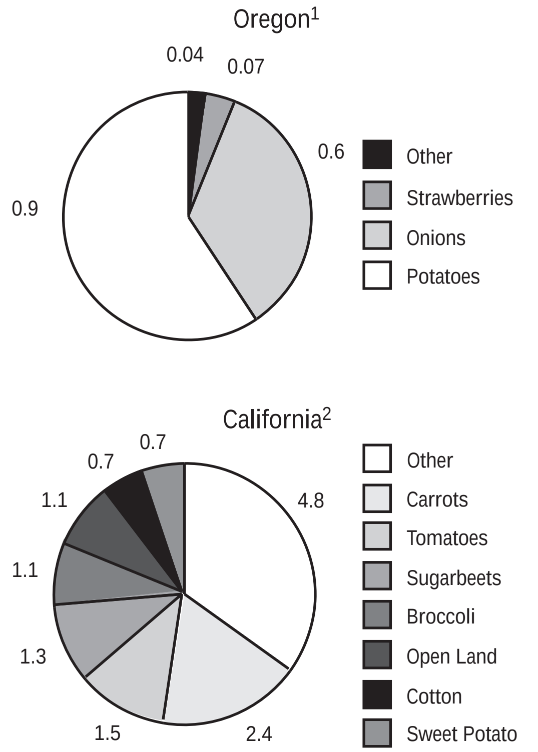
Carcinogenicity

Several kinds of cancer have been associated with exposure to 1,3-dichloropropene in humans and animals. In 1984, two doctors published reports of three patients with malignancies believed to be associated with exposure to 1,3-dichloropropene. Two were firefighters who assisted with the 1973 cleanup of a 1,3-dichloropropene spill from a jackknifed trailer truck in Los Angeles. Both were later diagnosed with malignant lymphoma and died in 1980. The third patient was a farmer who was exposed to the chemical in 1975 through a broken hose connection on his application equipment. He was diagnosed with leukemia about a year following exposure and died shortly thereafter.[14]

The National Toxicology Program (NTP) published results of carcinogenicity studies (studies of the ability of a chemical to cause cancer) of Telone II on mice and rats in 1985. Increases in a stomach cancer were found in male rats exposed to Telone II, while increases in a lung cancer were found in both male and female mice. In addition, exposure to Telone II caused tumors in the livers of male rats and in the stomachs and livers of female mice.[15] (In all of these studies Telone II was administered by gavage.) In addition, injection of cis-1,3-dichloropropene under the skin of mice caused an increase in the incidence of another cancer.[3] Based on these results, EPA has classified 1,3-dichloropropene as a category B_2 (possible human) carcinogen.[7] The World Health Organization has judged there to be "sufficient evidence" of 1,3-dichloropropene's carcinogenicity to experimental animals.[16]

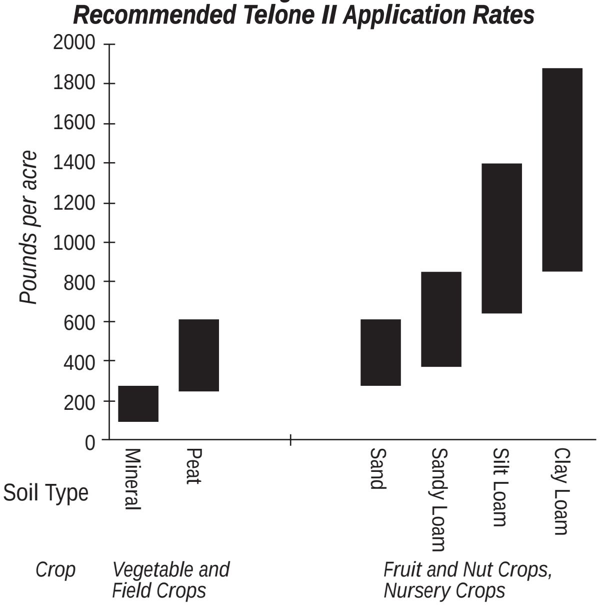
Based on the results of worker exposure studies conducted by Dow Chemical Company, EPA calculated cancer risks for a variety of people who might be exposed to 1,3-dichloropropene. The estimates ranged as high as almost six extra cancer cases per thousand workers exposed in storage areas and almost two cancers per thousand people exposed in fields immediately after treatment. The risk for people living downwind from treated fields was estimated at nearly five extra cancer cases per ten thousand people exposed.[3] (See Figure

Figure 2
Telone II Uses in 1987
(millions of pounds)



1. Rinehold, J.W. and J.M. Witt. 1989. *Oregon pesticide use estimates for 1987*. Special Report 843. Corvallis, OR: Oregon State University Extension Service.
2. "ARB monitoring for 1,3-dichloropropene." Memorandum from R.J. Oshima, California Department of Food and Agriculture Environmental Monitoring and Pest Management, to G. Shiroma, Air Resources Board, Toxic Air Contamination Branch. February 7, 1990.

Figure 3
Recommended Telone II Application Rates

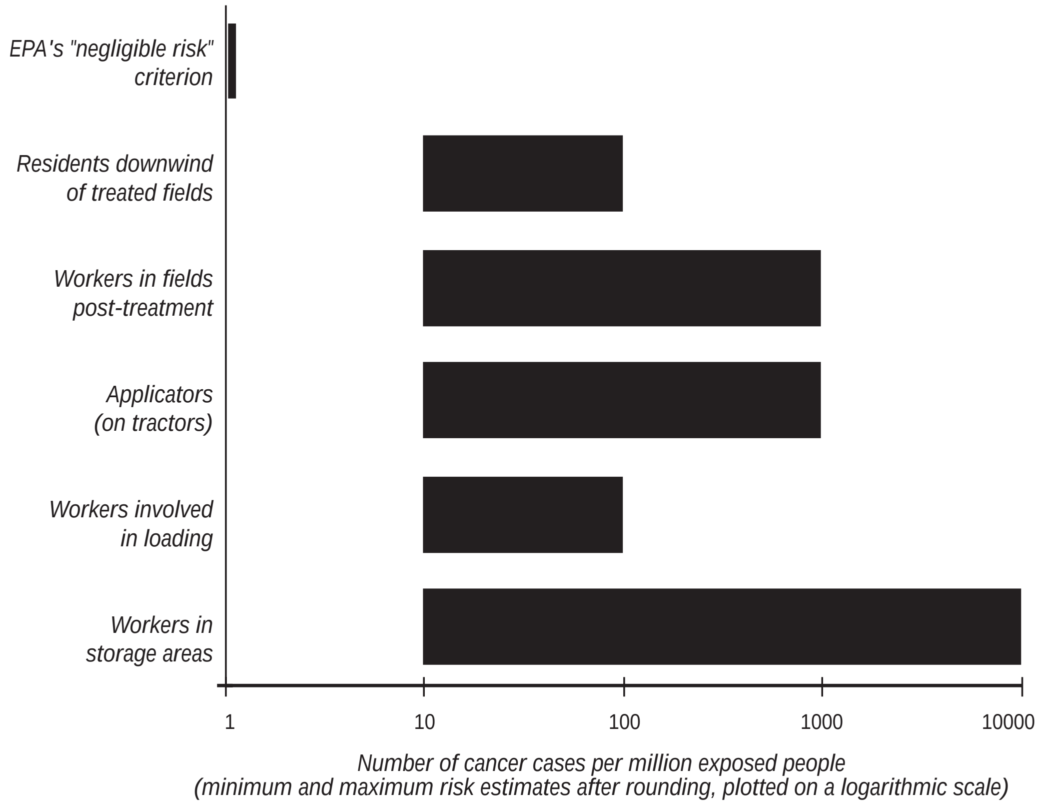


Source: DowElanco. 1991. *Specimen label: Telone II Soil Fumigant.* 6 pp. Indianapolis, IN.

4.)

NTP considered the possibility that some of Telone II's carcinogenicity resulted from ingredients other than 1,3-dichloropropene.[15] The formulation of Telone II tested by NTP contained two ingredients that are carcinogenic: about one percent of a stabilizer, epichlorohydrin, and about 2.5 percent of a manufacturing impurity, 1,2-dichloropropane. (See Figure 1.) Because 1,2-dichloropropane has less than one-tenth the carcinogenic potency of 1,3-dichloropropene[3] and is present in small amounts, NTP believes that the presence of the 1,2-dichloropropane impurity could not account for the carcinogenicity of Telone II. The epichlorohydrin is a much more potent carcinogen but it is "doubtful that Telone II contained enough epichlorohydrin for the tumor response to be due solely to epichlorohydrin."[7]

FIgure 4
EPA Estimates of Cancer Risks from Telone II Exposure



Source: U.S. EPA. Office of Pesticides and Toxic Substances. 1986. *Guidance for the reregistration of pesticide products containing 1,3-dichloropropene as the active ingredient.* Washington, D.C.

New formulations of Telone II contain an epoxidized soybean oil as a stabilizer instead of epichlorohydrin.[7] Carcinogenicity tests showed that inhalation of the new formulation caused increases in benign lung tumors in male mice; the formulation has not been tested using the gavage exposure used in the earlier studies of the epichlorohydrin formulation.[17]

Another impurity, a trichloropropene isomer accounting for about 1.5 percent of Telone II, has not been tested for carcinogenicity.[15]

Mutagenicity

1,3-Dichloropropene has been shown to cause mutations in a variety of laboratory tests. In fruitflies, 1,3-dichloropropene causes an increase in the number of sex-linked recessive lethal mutations.[18] In mammalian cells (including continuous-culture human cells, hamster ovary cells, and hamster lung cells) 1,3-dichloropropene, or the soybean-oil formulation of Telone II, caused four different kinds of mutagenic (damaging to genes) effects.[7] Ten different studies conducted between 1977 and 1988 found that 1,3-dichloropropene and its formulated products had mutagenic effects on bacterial cells.[7] In addition, 3-chloroallyl alcohol, a degradate of 1,3-dichloropropene in soils, has also been shown to be mutagenic to bacteria.[19]

Mutagenicity studies of cis-1,3-dichloropropene that has been chemically purified[20,21] have shown that the mutagenicity of the compound is likely due to the mutagenicity of impurities that arise during its manufacture and storage. These impurities appear to be oxiranes (see Figure 1).

Reproductive Effects

There is evidence from laboratory tests that exposure to 1,3-dichloropropene has some effects on reproduction. Rats that inhaled 300 parts per million (ppm) Telone II (with epichlorohydrin) during the middle of their pregnancies had fewer fetuses per litter and an increase in the number of fetal resorptions.[7] In another study, pregnant rats and rabbits that inhaled Telone II (with epichlorohydrin) during 9 (rat) or 12 (rabbit) days during the middle part of their pregnancies ate less food, drank less water and gained less weight than untreated animals. Bone formation in the vertebrae of the rat offspring was delayed at the highest exposure level (120 ppm).[22] Decreased maternal body weights, as well as damage to the lining of their noses, were noted in a two generation study of rats that inhaled the new Telone II formulation (with epoxidized soybean oil).[23]

Human Exposure

1,3-dichloropropene and its metabolites have been found in and around workers who are using the fumigant. Personal air samplers on commercial applicators in Dutch flower-bulb fields measured 1,3-dichloropropene in the air on all 21 person-days sampled. Concentrations that exceeded the Dutch occupational exposure limit (5 mg/m^3 or 1 part per billion, equal to the U.S. threshold limit value and the limits for air contaminants set by the Occupational Safety and Health Administration (OSHA))[7] were found during 30 percent of the observed working days.[24] In Hawaiian pineapple fields, personal air samplers detected 1,3-dichloropropene in all samples taken; one of the 72 workers was exposed to concentrations above the threshold limit value.[25] Concentrations of almost twice the OSHA limit were measured in a study of California Telone II applicators.[12]

Protective clothing is required for workers during handling and application of Telone II.[3] However, there are no materials that are "completely impervious to penetration by liquid Telone II" according to the manufacturer.[26]

The air monitoring studies in California show that exposure is not limited to workers; people living near agricultural areas where 1,3-dichloropropene is used as a fumigant are also exposed.

Ground and Surface Water Contamination

Tests for 1,3-dichloropropene residues in groundwater have detected the pesticide in five states: Connecticut, California,[27] Nebraska,[28] New Jersey, and Oregon.[27] No lifetime drinking water health advisory level has been established by EPA for 1,3-dichloropropene because it is carcinogenic.[29] However, concentrations of 1,3-dichloropropene in Connecticut and New Jersey were above a "surrogate" health advisory based on the kidney effects found in the rat gavage study mentioned above.[27]

These findings are consistent with 1,3-dichloropene's chemistry. Because it is highly soluble in water, does not adsorb well to soil, and does not evaporate readily when dissolved in water it is likely to leach through soil into groundwater.[7]

Although these chemical characteristics favor leaching, 1,3-dichloropropene is only moderately persistent. Half-lives (the length of time required for half of the initial amount applied to change into other compounds) have ranged between 3 and 69 days.[7] Therefore, it has not been found in ground water as often as its volume of use might predict, or as often as its more persistent chemical relatives. For example, an EPA survey estimated that the soil fumigant ethylene dibromide contaminates 19,200 wells in the U.S., but did not find any wells with 1,3-dichloropropene residues.[30]

Under certain conditions, however, it can persist in soil or groundwater much longer. For example, monitoring of bulb fields in the Netherlands prior to application of Telone II found small amounts that appeared to have persisted from applications three to four years earlier.[31] Groundwater under Dutch potato fields contained 1,3-dichloropropene at a depth of almost twenty feet six years after application.[32] In New York, 1,3-dichloropropene applied to potato fields was found in groundwater at a depth of about 10 feet for 138 days after application. Peak concentrations were not measured until 83 days after application.[33]

"Protective clothing is required for workers during handling and application of Telone II. However, there are no materials that are 'completely impervious to penetration by liquid Telone II' according to the manufacturer."

1,3-Dichloropropene has been found in the drinking water in New Orleans, Louisiana,[34] and in rainwater in Portland, Oregon.[35]

Effects on Nontarget Organisms

The aquatic toxicity of 1,3-dichloropropene has been little studied.[36] Juvenile sheepshead minnows are killed by concentrations of 1,3-dichloropropene in their water that exceed 1.2 parts per million (ppm). It was more toxic than all but twelve chemicals in a survey[37] of the toxicity to minnows of 54 industrial chemicals. The LC_{50} (the concentration that will kill 50 percent of a population of test animals) is 2.2 ppm for minnows exposed for 72 hours.[37] A similar LC_{50}, 0.5 ppm, (based on a fourteen day exposure) has been reported for freshwater guppies and for shrimp (0.8 ppm).[36] The water flea *Daphnia magna*,[5] the bluegill sunfish,[5] rainbow trout,[5] and golden orfe[34] all have LC_{50}s that are only slightly higher (48 or 96 hour LC_{50}s: 6.2,[5] 7.1,[5] 3.9,[5] and 9 ppm[34] respectively).

Nonlethal effects occur at lower concentrations. For example, the amount of chlorophyll and the number of cells in algae are reduced by concentrations of between 1 and 5 ppm of 1,3-dichloropropene.[36] Saltwater algae were most sensitive.[38] Chronic effects were found on embryos and larvae of fathead minnows at concentrations as low as 0.24 ppm, about one-tenth the LC_{50}.[38]

Fumigation with 1,3-dichloropropene may cause increases in the levels of ammonia in soils, particularly if they are cold, wet, acidic, or high in organic matter. Injury to crops can result.[26] This appears to be caused by an imbalance in the soil microflora; nitrifying bacteria are suppressed by the fumigation while ammonifying bacteria are not inhibited.[39] The ecology of mycorrhizal fungi is also affected by fumigation. Certain species are inhibited by 1,3-dichloropropene while spore formation in others is increased, probably because nematode and other parasites are killed by the fumigation.[40]

Regulatory History

1,3-Dichloropropene was first registered as a pesticide in 1966. Twenty years later EPA completed its review of health and environmental data submitted by the manufacturer for reregistration and published data requirements that had yet to be met.[3] At the same time EPA initiated a Special Review, the process by which it evaluates the risks and benefits of pesticides thought to pose unreasonable adverse effects, because of the concerns about its carcinogenicity. As of 1992, the Special Review is still in progress. Because registered uses of two other soil fumigants, EDB and DBCP, were cancelled during the 1980s because of concerns over their carcinogenicity, mutagenicity, and reproductive effects,[41] use of 1,3-dichloropropene has increased.[25]

Summary

1,3-Dichloropropene is an acutely and chronically toxic soil fumigant. By weight it is the fifth most abundantly used pesticide in the U.S. In humans, it causes respiratory problems, skin and eye irritation, and kidney damage. Kidneys appear to be particularly sensitive to the chemical. It causes cancer in laboratory animals and genetic damage in insects and mammal and bacteria cells. It leaches readily and has been found in U.S. groundwater, drinking water, and rainwater. Aquatic organisms are killed by concentrations of less than ten parts per million of 1,3-dichloropropene.

1,3-Dichloropropene applicators and agricultural workers in treated fields are occupationally exposed to the fumigant, as are those involved in the manufacture, transport, or disposal of the chemical. Consumers of the potatoes, carrots, strawberries, pineapples, and other crops that are grown after fumigation with 1,3-dichloropropene are in effect requiring these workers to expose themselves to this toxic chemical. It is clear evidence of the need for sustainable agricultural practices. ■

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