DCPA (Dacthal)

By Caroline Cox

Dimethyl tetrachloroterephthalate, commonly known as dacthal, DCPA, or chlorthal-dimethyl, is an herbicide active ingredient used to control crabgrasses, other annual grasses, and certain broad-leaved weeds in turf, home flower gardens, nursery stock, and a number of fruit and vegetable crops.[1] First registered for use in the United States in 1958, it is manufactured by the Fermenta Plant Protection Company and sold under the trade name Dacthal.[2] Estimated annual usage in 1988 was between 3.2 and 4.7 million pounds. Only 37 percent of this is used on crops; the majority is used on turf sod farms, golf courses, home lawns, and home gardens.[3] Estimated use on golf courses in 1982 was 400,000 pounds.[4]

DCPA is a chlorinated benzoic acid[2] (see Figure 1) in the same chemical family as the herbicides dicamba and chloramben.[5]

Mode of Action

DCPA kills germinating seeds. Microscopic studies indicate that it does this by disrupting microtubule formation in exposed cells and causing abnormal cell division. Microtubules are slender cylindrical structures made of polymerized proteins. They move chromosomes to newly forming daughter cells and control the orientation of cell walls in plant cells. Abnormal microtubules disrupt cell wall formation, resulting in the formation of large multinucleate cells when cell division is inhibited. They also disrupt the process by which chromosomes replicate and divide.[6,7]

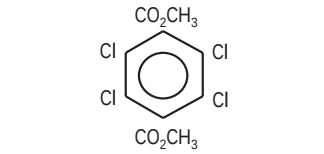
Acute Toxicity

Of the six acute toxicity tests required by the U.S. Environmental Protection Agency (EPA) for the registration of a pesticide, only one (a skin sensitization test in guinea pigs) had been completed at the time DCPA's registration standard was issued in 1988. The five missing tests include oral toxicity or LD_{50}* (in rats), dermal toxicity (in rabbits), inhalation toxicity (in rats), eye irritation, and dermal irritation (in rabbits).[3] Based on other EPA data, the acute oral LD_{50} appears to be greater than 10,000 milligrams per kilogram (mg/kg) of body weight in both rats and dogs.[8]

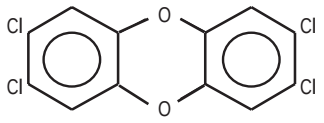
Chronic Toxicity

In a 1963 study, rats fed DCPA for two years had increased kidney weights (in males), increased adrenal gland weights (in females), excessive growth of the thyroid, abnormalities of the liver, and accumulation of iron in the spleen (in females). The No Observable Effect Level (NOEL) for these symptoms was 50 mg/kg/day.[2] EPA believes that the test was not adequate to detect all of the chronic effects of DCPA because a persistent lung infection was present in both treated and untreated animals, resulting in a high mortality rate. In addition, the sample size of the test was smaller than current EPA standards.[9] EPA is requiring that the test be repeated. NOELs are used by EPA and other regulatory agencies to establish permissible exposure levels to pesticides, and the results of this rat study are being used even though the study is inadequate. A chronic feeding study of dogs found no adverse effects,[3] but a subchronic (28 day) study found weight loss, decrease in liver weight and liver degeneration at the only dose tested, 800 mg/kg/day.[10]

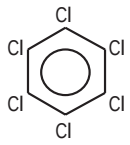
Figure 1
DCPA and Its Contaminants



DCPA



2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD)



Hexachlorobenzene (HCB)

Tests required for U.S. registration of dacthal found no teratogenic (causing birth defects) or mutagenic (causing genetic damage) effects,[2] although a Russian study of mouse bone marrow cells found that dacthal caused abnormalities in cell division.[11] Valid tests of DCPA's oncogenicity (ability to cause tumors) and reproductive toxicity had not been completed by 1988,[2] although the two-year rat study (above) found a variety of tumors.[9]

Contaminants and "Inert" (Secret) Ingredients

DCPA is contaminated by two toxic impurities, 2,3,7,8-tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD) and hexachlorobenzene (HCB).[3] (See Figure 1.) Levels of 2,3,7,8-TCDD in DCPA are as high as 100 parts per trillion, while HCB has been found at levels up to three parts per thousand.[3] Since both of these compounds are toxic, their presence in DCPA is of concern.

2,3,7,8-TCDD is the most toxic member of the dioxin family of chlorinated organic (carbon-containing) compounds. In animals, dioxins cause a variety of adverse health effects, including cancer, birth defects, atrophy of the thymus, liver damage, reduced functioning of the immune system, reproductive failure, skin disfiguration, and weight loss[12] (JPR 9(4):32-36).

Many of these effects are found following exposure to minute amounts of 2,3,7,8-TCDD. For example, both carcinogenic and reproductive effects have been found at levels in animals of one part per trillion.[13,14,15] Mortality, reduced growth, and lethargic behavior in rainbow trout was found in water containing 38 parts per quadrillion of 2,3,7,8-TCDD.[16]

2,3,7,8-TCDD persists in soil; estimates of its half-life in soil range from 10 to 30 years.[17] It also concentrates in exposed animals, particularly in fish. In the rainbow trout study mentioned above, bioconcentration factors in fish were cal-

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* The LD_{50} is the dose of a chemical that kills 50 percent of a population of test animals.

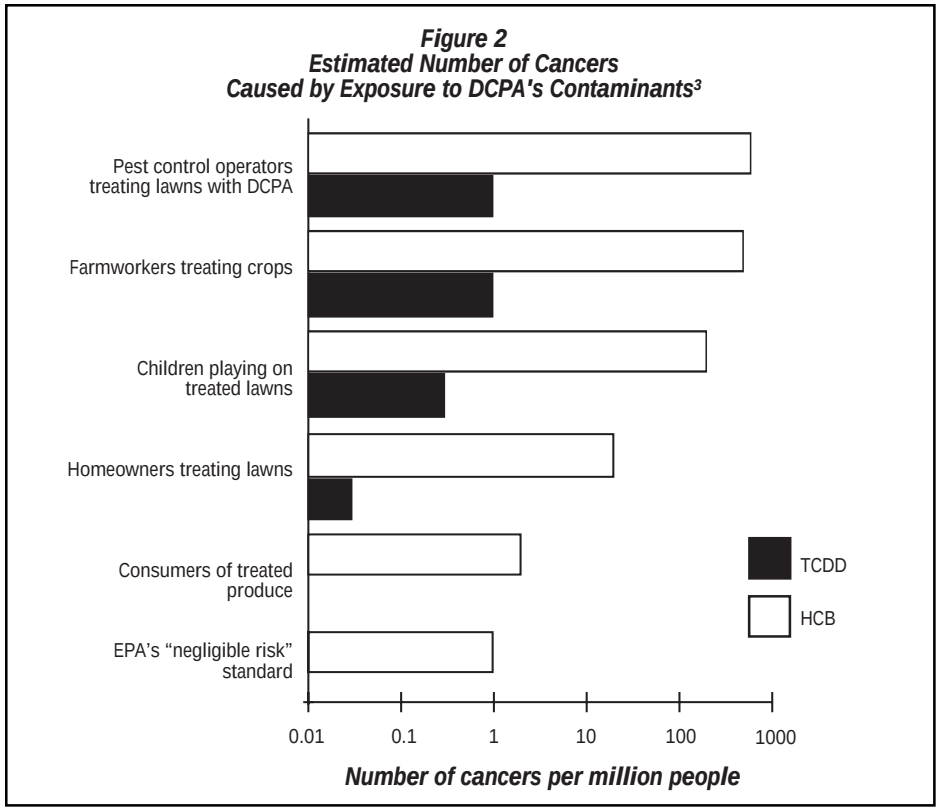


Figure 2
Estimated Number of Cancers
Caused by Exposure to DCPA's Contaminants[3]

culated to be over 28,000. Similarly large bioconcentration factors have been calculated for carp and fathead minnows. In cattle, 2,3,7,8-TCDD concentrations in beef and milk fat are approximately five times those found in the cattle feed.[17]

HCB has been associated with arthritis, osteoporosis of bones in the hands, liver damage (including the disease porphyria cutanea tarda), skin lesions, muscle weakness and short stature in humans.[18] HCB is concentrated in breast milk and high concentrations are passed from mother to child during nursing. In Turkey (where three to four thousand people consumed HCB contaminated bread in 1955) all infants born to mothers with porphyria died. Mortality of nursing babies was also high.[18] In animals, exposure to HCB causes similar effects. In addition, HCB in animals causes kidney damage, immune system suppression, neurologic effects, cleft palate, a reduction in the survival of nursing pups, and cancer.[18]

HCB residues have been found in the blood of agricultural workers using ground spray equipment to apply DCPA.[19] Nineteen of twenty workers studied had blood residues, and the concentration of HCB was correlated with the length of time the employees had worked with DCPA.[19]

HCB is relatively resistant to degradation, and its half-life in soil has been estimated between three and six years.[18] HCB bioconcentrates by a factor of 11 in chickens, over 6,000 in catfish, and 140,000 in seagull eggs.[20,21] DCPA contains almost ten times as much HCB as the other three pesticides contaminated with HCB (chlorothalonil, picloram, and pentachlorophenol). A total of about 12,500 kilograms of HCB per year are released into the air in the United States as these pesticides are used.[18]

EPA estimated the carcinogenic risks associated with the 2,3,7,8-TCDD and HCB contaminants in DCPA. The results are as high as six hundred cancers per million pest control operators exposed to HCB while treating lawns with DCPA and two hundred cancers per million children playing on DCPA-treated lawns.[3] (See Figure 2. For a discussion of the problems with quantitative risk assessment, see JPR 8(1):7-12 and JPR 10(1):2-7).)

The public has no information about the identity of the "inert" ingredients used in DCPA formulations or their toxicity, either alone or in combination with DCPA. According to pesticide manufacturers, the identity of these ingredients is a trade secret. EPA doesn't require testing of "inert" ingredients and requires only acute toxicity testing of the formulated products (active plus "inert" ingredients). A typical DCPA formulation, Dacthal G-25, contains 50 percent secret ingredients.[1]

Human Exposure

DCPA residues have been found in the Total Diet Study conducted by the U.S. Food and Drug Administration. The study samples a "market basket" of 234 food items collected four times a year from different regions of the U.S. and tests for pesticide residues. The samples are analyzed for over 200 chemicals. In 1987, DCPA was found 20 times in the 936 items sampled; it ranked twentieth in frequency out of the 53 pesticides found in the survey. HCB was found 91 times and ranked sixth in frequency.[22]

EPA has established a Provisional Acceptable Daily Intake for DCPA of .5 mg/kg/day based on the inadequate two year rat study discussed above.[2]

Environmental Fate

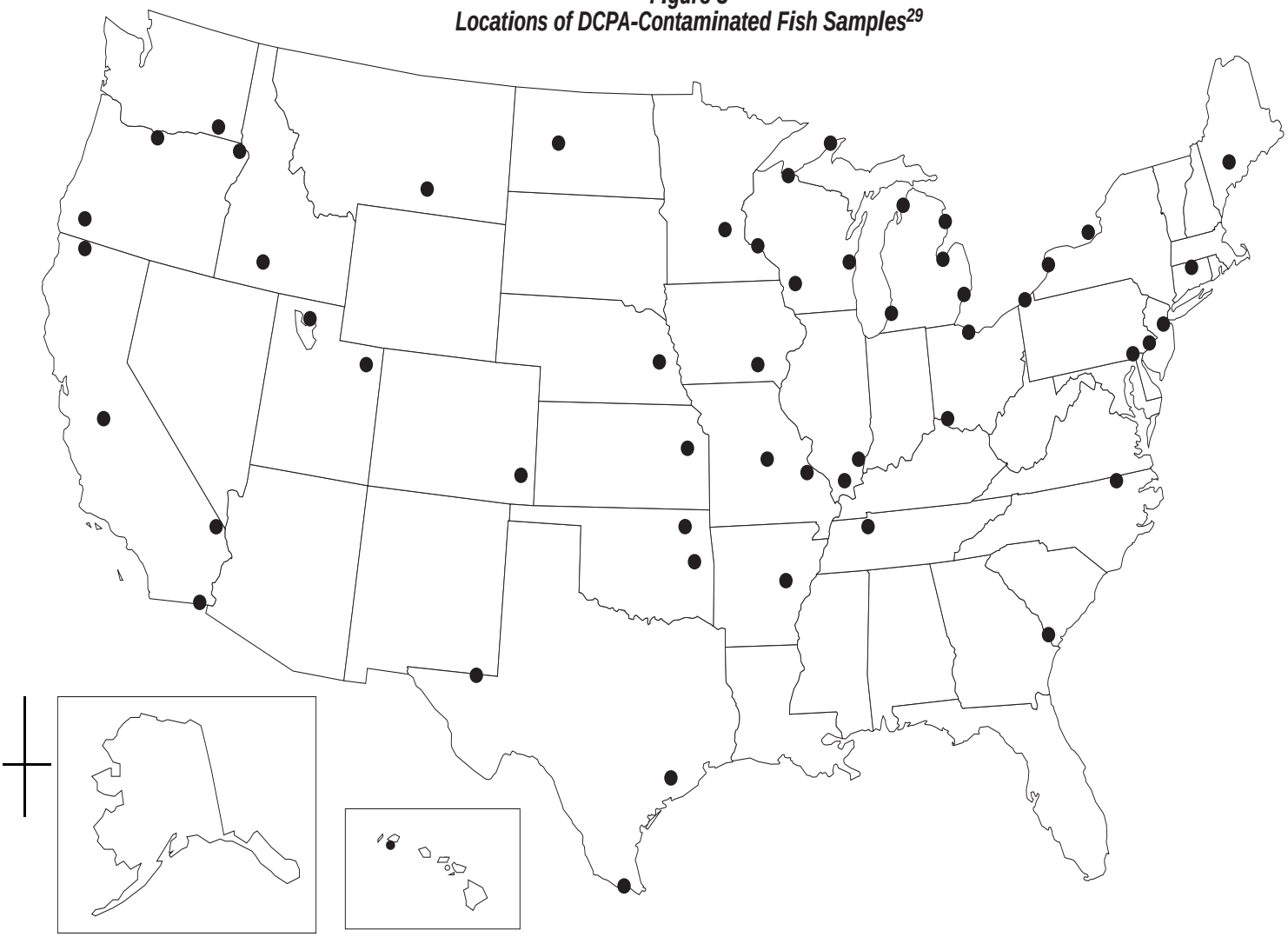
In 1988, nine of the ten environmental fate studies required by EPA for DCPA's registration were incomplete.[3]

Degradation of DCPA in soil is dependent on both temperature and soil moisture. Half-lives in a recent study varied from 18 days at 30° C to 92 days at 10° C. Low soil moisture (10 percent) increased DCPA's half-life.[23] Volatilization (evaporation) also increased with increasing temperature.[24]

Drift of DCPA onto parsley, daikon, dill, and kohlrabi (crops on which DCPA use is not registered) has been documented in California. A field study of DCPA drift to quantify this problem showed that DCPA volatilization continued up to 60 days after application. About 10 percent of the DCPA was lost to the atmosphere in the first 21 days after application. Half-life in the soil in this study was 50 days.[25]

DCPA and its metabolites (breakdown products which EPA considers toxicologically equivalent to DCPA[3]) have been repeatedly found in groundwater and surface water. In EPA's National Pesticide Survey, over 6 percent (about 6,000) of the community water system wells contained DCPA metabolites as did over 2 percent (about 264,000) of rural domestic wells. DCPA's contaminant HCB was found in 0.5 percent (about 470) community water system wells. DCPA metabolites were the most commonly detected pesticide residue in the survey. Since a larger percentage of community water system wells were contaminated by DCPA than were rural domestic wells, EPA believes that much of the contamination may have come from urban rather

Figure 3
Locations of DCPA-Contaminated Fish Samples[29]



than agricultural uses.[26] EPA's STORET water quality database indicated in 1988 that 19 percent of surface water samples (386/1,995) and one percent of ground water samples (12/982) were contaminated with DCPA.[8] The highest DCPA levels found in groundwater were 1,039 parts per billion (ppb) found in Suffolk County, New York[8] and 986 ppb found in Malheur County, Oregon.[27]

EPA has calculated a lifetime health advisory level for DCPA in drinking water of 4,000 micrograms per liter (equivalent to 4,000 ppb).[8] This level is based on the toxic effects found in the inadequate two year rat study discussed above, although the study is being replaced. Earlier guidance levels for DCPA in drinking water, though much lower (500[9] and 3500 ppb[28]), were based on the same study. The increase in the advisory levels has come, not from changes in the results of toxicity tests, but from changes in the factor used to calculate a margin of safety (from 1000 to 1,00) and changes in assumptions used to calculate the advisory levels.*

Effects on Nontarget Organisms

Like residues of other persistent organochlorine pesticides (DDT, toxaphene, pentachlorophenol, dieldrin, and chlordane, for example), residues of DCPA in fish are widespread. The U.S. Fish and Wildlife's National Contaminant Biomonitoring Program analyzed fish samples from 112 stations in rivers throughout the U.S. as well as the Great Lakes. DCPA residues were found in 34 percent of the samples from 1978-79, 28 percent of the samples from 1980-81, and 45 per-

* Looking closely at the calculation of these health advisory levels is an interesting exercise. The first (draft) level set in 1982 calculated a "longer-term" advisory level, one that is based on the water consumption and body weight of a child using the previously mentioned inadequate two year rat study. In the 1987 revision, the longer-term level was based, not on the two year rat study, but on a 28 day rat study (also judged inadequate) and the two year study was used to calculate a lifetime advisory level. Lifetime levels are based on the water consumption and body weight of an adult. This change, as well as the change in the assumed margin of safety (from 1000 to 100) resulted in an increase in the advisory level from 500 to 3500 ppb. The margin of safety is supposed to compensate for the inadequate tests, so it is ironic that only the margin of safety, and not the quality of the testing, were changed between 1982 and 1987. In 1988, the levels were rounded off to the nearest 1000 ppb, resulting in another increase to 4,000 ppb. All of the changes have come, not from new information about the toxicity of dacthal, but from new assumptions and calculations.

cent of the samples from 1984 (See Figure 3).[29] Highest concentrations were found in the lower Rio Grande (Texas) where agricultural use of DCPA has been widespread. HCB, which contaminates DCPA, was detected in 19 percent of the 1984 samples. Fish from four out of five rivers in the heavily agricultural San Joaquin Valley in California also contained DCPA residues.[30]

In Great Lakes fish, DCPA residues are even more common. In a 1980-81 survey, DCPA was detected in 73 percent of the samples, while HCB was found at all eight of the sites where collections were made.[31] At Siskiwit Lake, on Isle Royale in Lake Superior, DCPA residues were found in both lake trout and whitefish. Because of the lake's remote location in a national park where no pesticides have been used since some DDT applications were made in the 1960s, the authors of this study believe that the presence of DCPA indicates that the herbicide has been transported over long distances from the sites where it was applied and then deposited in Lake Siskiwit.[32] DCPA's volatility increases this kind of long distance transport.

Mussels growing in intertidal areas of the central California coast also contain DCPA residues. A 1982-83 survey found DCPA in mussels from eight out of the twelve sites sampled, including a site (Bodega Head) thought to be relatively uncontaminated with pesticides and PCBs.[33]

Summary

DCPA is a moderately persistent organochlorine herbicide that kills plants by inhibiting cell division. It has been registered for use in the United States since 1958, but most of the required testing is incomplete or invalid.

Chronic exposure to DCPA has caused adverse effects in the adrenal glands, kidneys, livers, thyroids and spleens of laboratory animals. DCPA is contaminated with two compounds, 2,3,7,8-TCDD and hexachlorobenzene, that are toxic (including carcinogenicity and reproductive abnormalities) at low doses. Both compounds bioaccumulate in animals. The chronic toxicity of commercial DCPA products cannot be assessed because the "inert" ingredients have not been tested.

Most Americans are unknowingly exposed to DCPA. It's metabolites are the most commonly detected pesticide residue in recent groundwater surveys and are often detected in surface water. DCPA is transported by air currents over long distances. Fish collected from sample locations throughout the United States are frequently contaminated by DCPA, with high concentrations in agricultural areas. ■

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