● HERBICIDE FACTSHEET

# PICLORAM

**The herbicide picloram (commonly sold under the trade names Tordon and Grazon) is typically used to kill unwanted broad-leaved plants on rangeland and pastures, in forestry, and along rights-of-way.**

**In laboratory tests, picloram causes damage to the liver, kidney, and spleen. Other adverse effects observed in laboratory tests include embryo loss in pregnant rabbits, and testicular atrophy in male rats. The combination of picloram and 2,4-D causes birth defects and decreases birth weights in mice.**

**Picloram is contaminated with the carcinogen hexachlorobenzene. Hexachlorobenzene, in addition to causing cancer of the liver, thyroid, and kidney, also damages bones, blood, the immune system, and the endocrine system. Nursing infants and unborn children are particularly at risk from hexachlorobenzene.**

**Picloram is toxic to juvenile fish at concentrations less than 1 part per million (ppm). Concentrations as low as 0.04 ppm have killed trout fry. In Montana, roadside spraying of Tordon killed 15,000 pounds of fish in a hatchery 1/4 mile downstream from the Tordon treatment.**

**Picloram is persistent and highly mobile in soil. It is widely found as a contaminant of groundwater and has also been found in streams and lakes. It is also extremely phytotoxic, and drift and runoff from picloram treatments have caused startling damage to crops, particularly tobacco and potatoes.**

**Because of these characteristics, both the Ecological Effects Branch and the Environmental Fate and Ground Water Branch of the U.S. Environmental Protection Agency (EPA) recommended that use of picloram not be continued. These recommendations were not accepted by EPA when it evaluated picloram in 1995.**

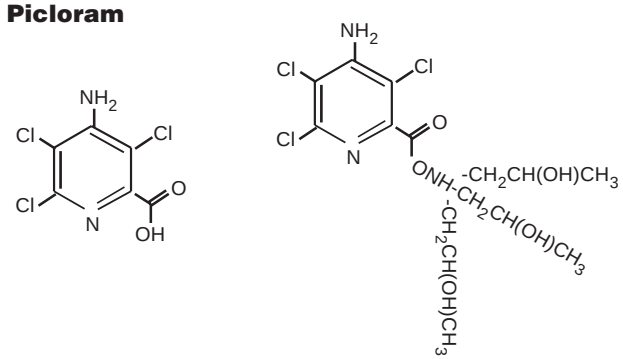
BY CAROLINE COX

Picloram is an herbicide in the pyridinecarboxylic acid family. It was first registered in the U.S. in 1964.[1] Picloram's primary manufacturer is Dow AgroSciences, and it is marketed under the brand names Tordon and Grazon.[2-6]

In 1995, picloram was reregistered by the U.S. Environmental Protection Agency (EPA), meaning that EPA had evaluated the health and safety testing submitted for picloram and found that it met current standards.[1] Picloram's registrations in California were withdrawn in 1986 because the manufacturer did not provide data about health effects and groundwater contamination required in California.[7]

Three forms of picloram are registered for use in herbicides. (See Figure 1.) Picloram acid is used only to manufacture other forms of picloram, the triisopropanolamine and potassium salts which are found in picloram herbicides.[1] A fourth form, isooctyl picloram, no longer has active registrations.[8]

**Figure 1**
**Picloram**




## Uses

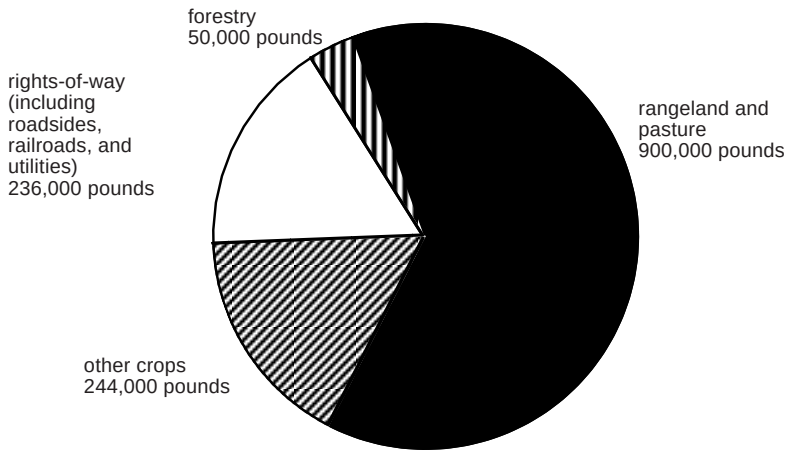
Picloram is used to kill unwanted broad-leaved plants on pastures and rangeland, in reforestation programs; in uncultivated areas; and along rights-of way. According to EPA estimates, its major use is on pasture and rangeland. (See Figure 2.) Between 1.4 and 2.1 million pounds are used annually in the U.S.[1,9]

## Mode of Action

Although herbicides that share a mode

**Caroline Cox** is JPR's editor.

**Figure 2**
**Estimated Annual Use of Picloram in the U.S.**




U.S. EPA. Prevention, Pesticides, and Toxic Substances. 1995. Reregistration eligibility decision (RED): Picloram. Washington, D.C., Aug.

Over 60 percent of the picloram used in the U.S. is used on rangeland and pastures.

of action with picloram have been in use for more then 50 years, their precise mode of action remains unclear.[10] In general terms, picloram kills plants by acting like auxins, plant growth hormones. It is more persistent than auxins, and inhibits the enzymes that normally break down auxins. This means it disrupts normal growth, causing abnormal stimulation and maturation of tissues. Plant growth then stops, and the roots of the plants deteriorate. This results in death.[11]

Most broad-leaved plants are susceptible to picloram, while most grasses are resistant. Susceptible species absorb more picloram than resistant ones, allow it to accumulate in meristematic (growing) tissue, and only slowly metabolize it into water soluble compounds.[12]

## Acute Toxicity

Tests submitted by the manufacturer to support picloram's registration found that all three forms of picloram are of low acute toxicity.[1] However, picloram is more toxic in other tests. The oral median lethal dose for the potassium salt in an experiment conducted by an EPA researcher was about five times more toxic than the manufacturer's data, 690 milligrams per kilogram (mg/kg) for female rats and 950 mg/kg for males.[13] Picloram is also more toxic via inhalation; EPA classified picloram acid in Category I (the most toxic category) for inhalation toxicity, and both the potassium and triisopropanolamine salts are in Category II.[1]

## Eye Hazards

Picloram herbicides are hazardous to the eye. Tordon K and Tordon 22K cause "substantial but temporary eye injury." Tordon RTU, Tordon 101, and Grazon P+D cause "eye irritation."[2-6]

## Effects on the Immune System

Picloram and both of the picloram salts are labelled as skin sensitizers by EPA.[1] This means that an initial skin exposure can cause a more serious reaction to subsequent exposures. Skin sensitization has also been observed in humans.[11]

## Subchronic Toxicity

Subchronic toxicity refers to toxic effects found after exposures of several weeks or months. Subchronic effects of picloram have been found in the liver, kidney, spleen, and skin.

In a 90-day feeding study of rats, picloram acid caused liver weight changes at three of the five doses tested.[1] In a six month feeding study of dogs, picloram acid caused decreased liver weights and decreased body weight gain at the highest dose tested.[1] In a 13-week rat feeding study of the triisopropanolamine salt, abnormal growth of liver cells was found at the two highest doses tested, and increased liver and kidney weights were found in females at the highest dose.[1] A 21-day mouse study found that picloram increased the weight of the spleen.[14] A 90-day study of rats who drank water contaminated with the potassium salt found dose-dependent mortality, and an exacerbation of kidney and liver lesions.[13]

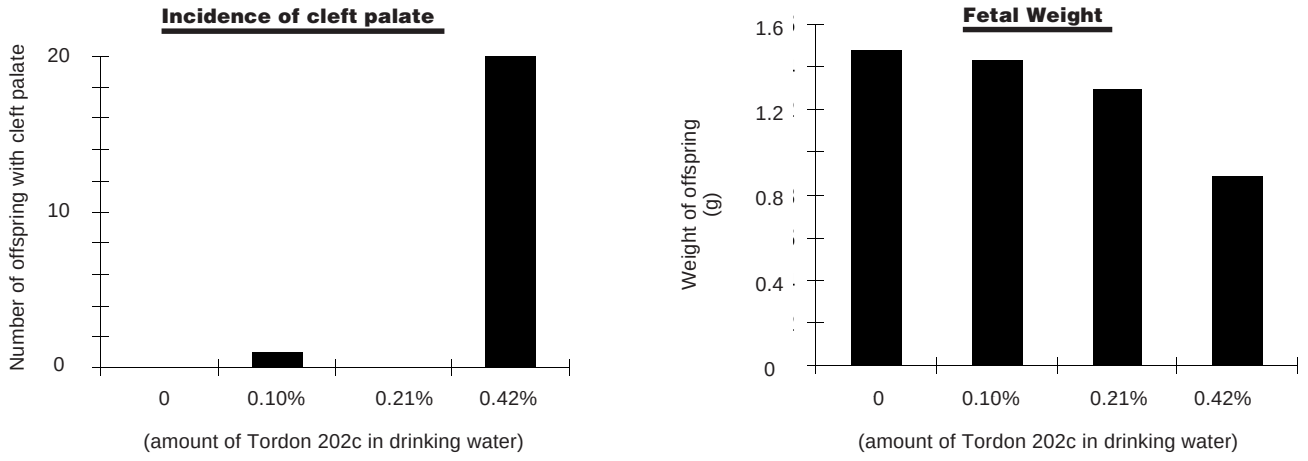
In tests of subchronic (21-day) dermal (skin) exposure in rabbits, picloram caused swelling and redness at every dose level tested. The tests were done using both the potassium and the triisopropanolamine salt.[1]

## Effects on Reproduction

Tests of the triisopropanolamine salt of picloram that were submitted by Dow in support of its registration found no effects on reproduction in rats and an increased rate of miscarriages at the highest dose tested in rabbits.[1] Tests of the potassium salt found increases in the frequency of embryo loss. Also, the frequency of umbilical hernias increased at all doses tested and multiple skeletal effects were increased at both the low and high dose tested.[15] Also, in male rats, feeding of picloram resulted in an increased frequency of atrophied testicles.[16]

Serious effects of picloram occurred in the offspring of pregnant mice who drank water contaminated with Tordon 202c. Like several Tordon and Grazon products, this herbicide contains picloram and the phenoxy herbicide 2,4-D. At the middle and high doses, fetal weight was reduced. The number of dead fetuses, the size of the fetuses, and the weight of the placenta were reduced at the highest dose. In addition, the incidence of cleft palate, a birth defect, increased dramatically at the highest dose tested.[17] (See Figure 3.)

**Figure 3**
**Reproductive Effects of Tordon 202c**




Source: Blakley, P.M., J.S. Kim, and G.D. Firneisz. 1989. Effects of gestational exposure to Tordon 202c on fetal growth and development in CD-1 mice. *J. Toxicol. Environ. Health* 28:309-316.

Tordon 202c, a mixture of picloram and 2,4-D, causes significant reproductive problems, including an increase in the frequency of cleft palate and a decrease in fetal weight.

Similar decreases in fetal size and weight, along with increased incidence of cleft palate, were found in a follow-up experiment in which male mice drank Tordon-contaminated water prior to conception of their offspring. In addition, malformations of the testes were more frequent, but the frequency did not consistently increase with dose.[18] A third study in which female mice drank Tordon-contaminated water, both prior to conception and during pregnancy, found similar results. Fetal size and weight, as well as placenta weight, were reduced at all doses tested. The incidence of cleft palate also increased, similar to the results of the first experiment.[19]

The authors of these studies give two possible explanation of why their tests found more serious effects on reproduction than other picloram studies. First, the combination of the two herbicides may be more toxic than either herbicide alone. Second, the reproductive problems may be a result of the so-called "inert" ingredients or contaminants found in the herbicides. (See "Contaminants," and "Inert Ingredients, p.16.) Because the Tordon 202c studies are the only publicly available studies of reproductive effects caused by commercial picloram-containing products, there is no way to decide which explanation is correct.

## Mutagenicity

Tests of picloram's mutagenicity, its ability to cause genetic damage, which were submitted by Dow AgroSciences in support of picloram's registration are negative.[1] However, a study conducted by the National Toxicology Program had different results. Chromosome aberrations increased in frequency in hamster ovary cells exposed to picloram. The frequency of sister chromatid exchanges (SCEs) also increased.[15] (SCEs are exchanges of genetic material during cell division between members of a chromosome pair. They result from point mutations.)

## Carcinogenicity

Federal and international agencies that have evaluated picloram's ability to cause cancer have come to different conclusions.

EPA considers the primary cancer risk from picloram exposure to come from hexachlorobenzene (HCB; see Figure 4) Hexachlorobenzene contaminates picloram during its manufacture; as part of picloram's reregistration, concentrations of HCB were certified by its manufacturer to be no more than 100 parts per million (ppm).[1] HCB is a "probable human carcinogen"[20] and has caused liver, thyroid, and kidney tumors in laboratory tests.[20] It is also a contaminant in the commonly used fungicide chlorothalonil and the herbicide DCPA (Dacthal).[21]

According to EPA's assessment, most dietary exposure to HCB-contaminated picloram comes from eating beef or drinking milk from cattle which grazed on picloram-treated pasture and range. EPA estimates that the cancer risk from HCB-contaminated picloram totals about 70 percent of the level EPA considers acceptable. They also note that HCB "occurs as an impurity in several other pesticide technical products, so overall dietary exposure to HCB is likely to be appreciably higher than HCB considered simply as a picloram impurity as considered in this analysis."[1] The risk estimate also does not include exposure through contamination of water, air, or through contact with contaminated surfaces. In other words, it would not be difficult for the

total pesticide-related cancer risk from HCB to exceed EPA's standard.

The National Toxicology Program (NTP) has also evaluated the carcinogenicity of picloram. Unlike EPA, this agency conducts its own laboratory tests. NTP found that feeding of picloram increased the frequency of liver tumors in female mice.[22]

According to the International Agency for Research on Cancer, there is "limited evidence" for the carcinogenicity of picloram in laboratory tests. They report that picloram increased the frequency of liver tumors in rats and increased the frequency of thyroid tumors in female rats.[23]

## Contaminants

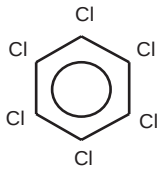
Hexachlorobenzene (see Figure 4) contaminates picloram, as well as other pesticides. Breathing hexachlorobenzene-contaminated air can harm the immune system, while ingesting hexachlorobenzene causes the liver disease porphyria cutanea tarda. Long-term feeding studies show that hexachlorobenzene harms the liver, thyroid, and nervous system, with additional damage to bones, kidneys, blood, the immune system, and the endocrine system. Hexachlorobenzene causes cancer of the liver, thyroid, and kidney in laboratory tests. Nursing infants and unborn children are particularly at risk from hexachlorobenzene because it is transferred from their mothers during pregnancy and nursing. Hexachlorobenzene is persistent, with half-lives (the amount of time required for half of the initial amount of a chemical to break down or dissipate) up to 6 years in soil and surface water, and up to 11 years in groundwater.[21]

## "Inert" Ingredients

All picloram-containing herbicide products contain so-called "inert" ingredients, ingredients added to the herbicide product to make it more potent and easier to use. The identity of some of these ingredients is publicly available. According to material safety data sheets produced by Dow AgroSciences, "inert" ingredients in Tordon and Grazon products include the following:

• **Ethylene glycol** (found in Tordon RTU[5]) can irritate the eyes, nose, and throat, and cause nausea, vomiting, and headache. Repeated or high exposure can damage the kidney and the brain. It can damage a developing fetus, and has been shown to cause birth defects in laboratory animals.[24]

**Figure 4**
**Hexachlorobenzene**




• **Triisopropanolamine** (found in Tordon RTU,[5] Tordon 101,[4] and Grazon P+D[6]) is a severe eye irritant. It also can cause skin irritation, nausea, vomiting, and respiratory tract irritation. Inhalation can be fatal because of spasms, inflammation, and fluid accumulation in the lungs.[25]

• **Isopropanol** (found in Tordon 101[4] and Grazon P+D[6]) is also known as isopropyl alcohol. Commonly used as a household disinfectant, it can irritate and burn the skin and eyes and irritate the nose and throat if inhaled. Overexposure can cause headache, drowsiness, unconsciousness, and death.[26]

• **Polyglycol 26-2** (found in Tordon K[3] and Tordon 22K[2]) is a proprietary surfactant and a complex polymer. Little toxicological information is publicly available about this chemical.

## Synergistic Effects

Two chemicals are said to be synergistic if the effect of a combination of the two chemicals is greater than the sum of the effects of the individual chemicals. Picloram is synergistic with several common herbicides with respect to its toxicity to mammals and fish. Picloram in combination with atrazine and alachlor causes liver toxicity and stimulates enzymes in the liver that are responsible for breaking down toxins.[14] As mentioned above, picloram and 2,4-D are synergistic in their negative reproductive impacts.[17-19] In livestock, the combination of 2,4-D and picloram has acted synergistically in causing mortality,[27] as well as in causing cancer of the small intestine.[28] Picloram and 2,4-D are also synergistic in their acute toxicity to trout.[29]

## Occupational Hazards

EPA has estimated exposure to hexachlorobenzene for workers who mix and apply picloram-containing herbicides. The HCB exposure of workers who apply picloram via backpack sprayers or low pressure handwands[1] exceeds the minimal risk level set by the U.S. Public Health Service for intermediate-term exposures to HCB.[21] The minimal risk level is "an estimate of the daily human exposure to a hazardous substance that is likely to be without appreciable risk of noncancer health effects."[21] The exposure estimates also exceed EPA's acceptable risk level for cancer over 10-fold.[1]

## Hazards to Fish

According to EPA, the picloram salts are slightly to moderately toxic to freshwater fish. Concentrations of 25 ppm of the triisopropanolamine salt kill rainbow trout, 20 ppm kill coho salmon, 24 ppm of the potassium salt kill bluegill, and 13 ppm kill rainbow trout.[1]

However, picloram is toxic to juvenile fish at much lower concentrations. Tests with the early life-stages of rainbow trout showed that concentrations of 0.9 ppm reduced the length and weight of rainbow trout larvae, and concentrations of 2 ppm reduced survival of the larvae.[30] A study of lake trout found that picloram reduced fry survival, weight, and length at the lowest concentration tested, 0.04 ppm.[31] A study of cutthroat trout used fluctuating concentrations designed to simulate field concentrations found in streams following picloram treatment of surrounding areas. Picloram concentrations are highest immediately after rain, then decrease until the next rain when

they increase again. Fluctuating concentrations with a maximum of 0.8 ppm reduced weight and length of trout fry. Unexposed fry had survival rates three times those of fry exposed to concentrations of picloram with a maximum of 1.6 ppm.[32]

Picloram-containing herbicides also cause serious sublethal effects in older fish. Yearling coho salmon exposed to 5 ppm of Tordon 22K for 6 days suffered "extensive degenerative changes" in the liver and wrinkling of cells in the gills.[33]

An incident near Sheridan, Montana, highlights the hazards of Tordon to fish.[34,35] In 1986,[36] and again twice in July, 1989,[35] a county roadside crew[35] sprayed about 1/4 mile upstream from a fish hatchery.[34] In both years,[36] rain fell within a few days of the spraying, washing Tordon 22K downstream and killing trout.[34,35] In the 1989 incident, fish turned black, became blind, and then died.[35] Eventually, 15,000 pounds of fish, all of the fish in the hatchery, were killed or left commercially unusable.[35]

## Hazards to other Aquatic Animals

The triisopropanolamine salt of picloram interferes with oyster shell formation at concentrations between 10 and 18 ppm. The potassium salt is toxic to oyster larvae at concentrations between 18 and 32 ppm.[1]

Epizootics (unusually high frequencies) of gonad tumors have been recorded in Maine softshell clams exposed to runoff from forestry and agricultural herbicides containing picloram.[37,38]

## Hazards to Nontarget Plants

Picloram, according to EPA is characterized by "extreme phytotoxicity." This of course is part of the reason for its commercial success. It also means that drift or runoff present serious hazards to nontarget plants. EPA evaluated the hazards to nontarget plants by calculating a "risk quotient" (RQ). RQs greater than 1 indicate what EPA calls "substantial risk." The RQ is calculated by dividing an estimate of exposure by an estimate of toxicity. The exposure values are based on estimates of drift and/or runoff, depending on the type of application. The RQs for picloram are extraordinary: for the potassium salt they range from 280 to 13,000 depending on the type of application.[1] (See Figure 5.)

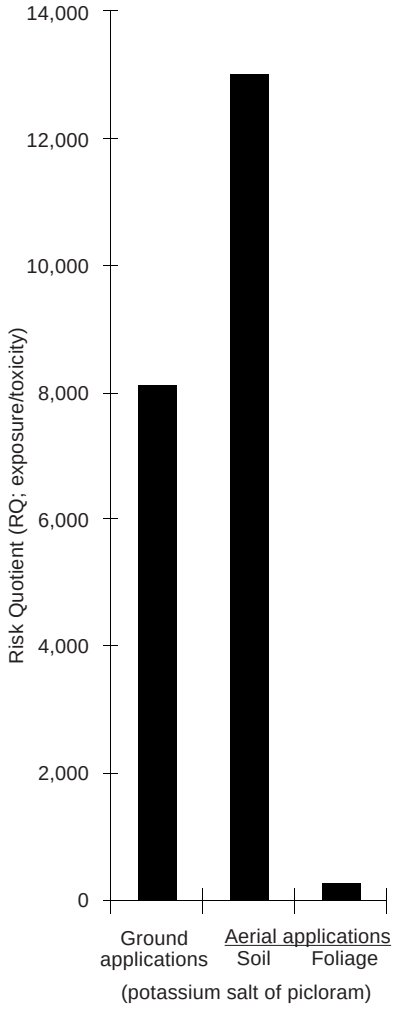
However, EPA believes these RQs *underestimate* actual risks because no assessment was done of risks at sites distant from the picloram application site. Plants at these sites could be exposed via irrigation with contaminated ground or surface water. "Effects at distant locations are plausible in view of the high persistence, mobility, and phytotoxicity of these chemicals," concluded EPA.[1]

EPA's Ecological Effects Branch (EEB) calculated that in order to reduce these risks below the "substantial" level, maximum use rates would have to be reduced to 0.0003 pounds per acre for the triisopropanolamine salt, and 0.0000473 pounds per acre for the potassium salt.[34] Since current use rates are typically 0.5 - 1 pounds per acre,[1] EEB felt that "practical mitigative measures cannot be identified"[34] for picloram. EEB "strongly"[34] recommended against the reregistration of all picloram products,[34] but its recommendation was not accepted by EPA's Reregistration Branch.[1]

Experimental support for EPA's calculations of the hazards of picloram drift or runoff comes from two studies. The first, looking at damage to tobacco caused by simulated runoff of picloram-contaminated water, found that the equivalent of 0.0002 pounds per acre of picloram reduced yields of tobacco.[39] In the second experiment, simulated drift of .05 pounds per acre reduced yields of cotton.[40]

Picloram's extreme phytotoxicity is also well illustrated by alarming incidents that have occurred in the 35 years since it was first commercially marketed. For example, mules were used to cultivate a tobacco field after they had grazed on a picloram-treated pasture. Picloram leached from their feces while they were working in the tobacco field, resulting in an "unusual spotty distribution" of stunted tobacco plants in the field.[41]

**Figure 5**
**Picloram's Hazards to Nontarget Plants**

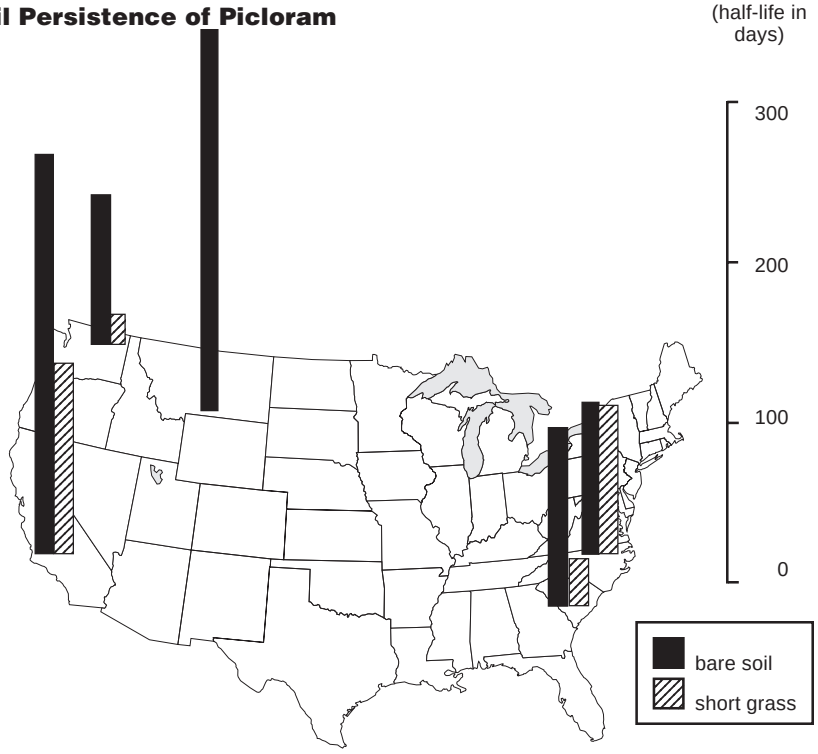



U.S. EPA. Prevention, Pesticides, and Toxic Substances. 1995. Reregistration eligibility decision (RED): Picloram. Washington, D.C., Aug.

EPA's estimates of picloram's hazards to nontarget plants are high, up to 13,000 times EPA's substantial risk level (RQ=1).

Another tobacco field was damaged by picloram following treatment of a utility right-of-way. Runoff from the right-of-way contaminated ponds that served as a irrigation water source for the tobacco fields. About 20 hectares (50 acres) of tobacco were injured, and an irrigation intake 4 kilometers (2.4 miles) from the treatment was contaminated.[39]

**Figure 6**
**Soil Persistence of Picloram**




Sources:
U.S. EPA. Office of Pesticide Programs. Environmental Fate and Ground Water Branch. Undated. Environmental Fate and Ground Water Branch review of picloram. Washington, D.C.
Cryer, S.A. et al. 1992. Picloram movement and dissipation in a northern rangeland environment. *Western Soc. Weed Sci.* 45:120-121.

Picloram's half-life in soil is typically over 100 days.

In Oregon, a roadside application of picloram contaminated a pond used to irrigate seed potatoes. The result was damage to 84 acres of potatoes, and a complete financial loss because the potatoes were unsalable as either seed or processing potatoes.[42]

Picloram also has other kinds of effects on plants. It can induce an increase in the frequency of chromosome aberrations in plant cells.[43] It also has inhibited nitrification in soil samples. Nitrification is the process by which ammonia is converted into nitrite and nitrate, and is the second stage of the nitrogen cycle in soil.[44] Plants depend on this cycle for usable forms of this essential nutrient.

**Aquatic plants:** EPA has been unable to assess picloram's hazards to aquatic plants because data for only one species were submitted as part of the registration process. Other data, however, indicates that hazards to aquatic plants are of concern. The aquatic plant *Myriophyllum sibiricum*, an important component of prairie wetlands whose fruits are eaten by waterfowl, was injured at a concentration of 0.01 ppm. A concentration of 0.1 ppm inhibited flowering. The researchers stated that because of the severity of injuries (at 0.1 ppm) "at least half, and perhaps all, of each population might have been incapable of producing viable propagules."[45] In addition, two species of algae in the genus *Hormidium* are killed by concentrations of picloram between 1 and 2 ppm.[46]

## Resistance of Weeds to Picloram

Yellow starthistle (*Centaurea solstitialis*) developed resistance to picloram in a Dayton, Washington, pasture that had been treated with the herbicide over a ten-year period.[47] Wild mustard (*Sinapis arvensis*) resistant to picloram was found in a field in Manitoba, Canada, that had been treated with herbicides that have a mode of action similar to picloram (dicamba, MCPA, and mecoprop) over a ten year period.[48] This cross-resistance, resistance to one herbicide conferring resistance to another herbicide, means that picloram-resistant weeds could be found in areas where picloram has never been used.

## Persistence in Soil

According to EPA, picloram is "resistant to biotic and abiotic degradation processes."[49] In other words, it is persistent in the environment because it is not easily broken down. "In some soils," continues EPA, "it is nearly recalcitrant to all degradation processes."[49] This recalcitrance is demonstrated by experimental calculations of its half-life, the length of time required for half of an applied amount of picloram to break down or move away from the application site. While the half-live under certain conditions can be as short as 21 days, in most cases the estimated half-life is over 100 days, and can be as long as 278 days.[49-50] (See Figure 6.)

Picloram's recalcitrance to degradation is also demonstrated by measurements that have been made of the time required for picloram to be completely gone from soil. In seven different studies located throughout the United States, picloram was still present between 1 and 3 years after treatment. In almost all of these studies, picloram was detected until the last sampling date, so that these are minimum estimates of persistence.[49-52]

Because of picloram's persistence, and its mobility in soil (see "Mobility in Soil," below), EPA's Environmental Fate and Ground Water Branch recommended that "picloram should not be reregistered because its use would pose unreasonable adverse effects to the environment."[49] However, this recommendation was not accepted by the Reregistration Branch.[1]

## Application Methods Causing Soil Contamination

Broadcast applications are probably the most common routes for picloram to contaminate soil. However, other more targeted application methods do have the potential to cause soil contamination. A study of leafy spurge found soil contamination after use of a "pipe-wick" applicator, a wiper-type applicator used to minimize contact of herbicides with the soil. The leaves took up the picloram, it was translocated to the roots and then released to the soil. Soil contamination was measured a week after treatment.[53] Similar root release has been measured in sweetgum and silver maple.[54]

## Mobility in Soil

EPA characterized picloram's ability to move through soil profiles in strong language. Picloram acid and its salts are "highly soluble"[1] in water, with the potassium salt having the highest solubility (740,000 ppm).[1] This means that picloram is "extremely mobile under field conditions"[1]; in fact, it is "among the most mobile of currently registered pesticides."[1] In field studies, it often leaches to the deepest part of the soil profile sampled.[1]

A good example of picloram's soil mobility comes from a study conducted in Arkansas. In one soil (a loamy fine sand)[55] virtually none of the picloram in experimental soil samples degraded, but nearly 100 percent of it leached.[1]

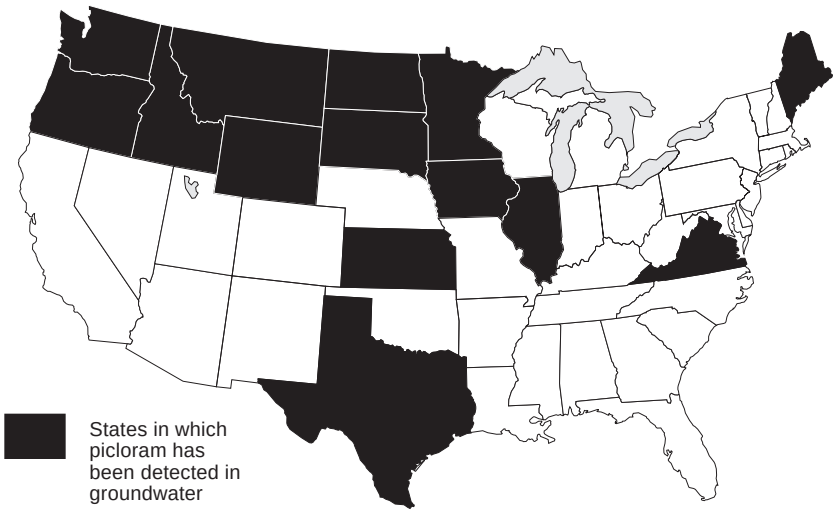
EPA's Environmental Fate and Ground Water Branch summarized their concerns about picloram's mobility in soil this way: "No practical use restriction can prevent it from contaminating the environment surrounding the target site."[49]

## Contamination of Groundwater

Because picloram is highly mobile in soil, it is likely to contaminate groundwater. EPA's evaluation states, "eventual contamination of groundwater is virtually certain in areas where residues persist in the overlying soil. Once in groundwater, the chemical is unlikely to degrade even over a period of several years."[1] Their evaluation is supported by groundwater monitoring studies. Picloram has been found in the groundwater of 14 states,[56-61] (see Figure 6) and also in Ontario and Saskatchewan, Canada.[62-63]

Groundwater aquifers are particularly susceptible to picloram contamination from roadside spray programs.[64] Most roadsides have ditches that parallel the road, and these ditches typically have had about a foot of soil removed, leaving very shallow soil profiles over aquifers. In addition, the extra runoff from the road surface increases leaching of picloram. Experimental herbicide treatments showed that "large amounts of the picloram … were found to leach through shallow road ditch soils and into the underlying aquifer material."[64]

**Figure 7**
**Picloram Contamination of Groundwater**




Source:
U.S. EPA. Office of Pesticide Programs. Environmental Fate and Ground Water Branch. Undated. Environmental Fate and Ground Water Branch review of picloram. Washington, D.C.
Cryer, S.A. et al. 1992. Picloram movement and dissipation in a northern rangeland environment. *Western Soc. Weed Sci.* 45:120-121.
Johnsen, T.N. 1980. Picloram in water and soil from a semiarid pinyon-juniper watershed. *J. Environ. Qual.* 9(4):601-605.
Michael, J.L., D.G. Neary, and J.M Wells. 1989. Picloram movement in soil solution and streamflow from a coastal plain forest. *J. Environ. Qual.* 18:89-95.

Picloram has contaminated groundwater in 14 states.

## Contamination of Surface Water

Picloram also easily contaminates surface water. For example, picloram was found for up to 275 days in West Virginia streams following hand application of Tordon pellets around the base of multiflora rose growing in pastures.[65] In Saskatchewan, picloram was found in a creek up to 35 months after a helicopter application of picloram granules. Following this same application, picloram was also found in a lake 1 kilometer from the treatment site.[63] Picloram was found in the Souris River, North Dakota, 1.5 kilometers from a wildlife refuge that had been treated with picloram for several years.[66] Picloram has also been found in streams or lakes in Alabama, Montana, North Carolina, Oregon, Texas, and Wyoming.[67]

## References

1. U.S. EPA. Prevention, Pesticides, and Toxic Substances. 1995. Reregistration eligibility decision (RED): Picloram. Washington, D.C., Aug.
2. Dow AgroSciences. 1998. Material safety data sheet: Tordon 22K Herbicide. Indianapolis IN, Jan. 1

3. Dow AgroSciences. 1998. Material safety data sheet: Tordon K Herbicide. Indianapolis IN, Jan. 1
4. Dow AgroSciences. 1998. Material safety data sheet: Tordon 101 Mixture Herbicide. Indianapolis IN, Jan. 1
5. Dow AgroSciences. 1998. Material safety data sheet: Tordon (R) RTU Herbicide. Indianapolis IN, Jan. 1
6. Dow AgroSciences. 1998. Material safety data sheet: Grazon P + D Herbicide. Indianapolis IN, Jan. 1
7. Letter from Duane Schnabel, registration specialist, Calif. Dept. of Pesticide Regulation, to Ellen Breiter, Apr. 15, 1996.
8. U.S. EPA. Office of Pesticide Programs. 1998. Chemical ingredients database. Maintained by Calif. Dept. of Pesticide Regulation. http://www.cdpr.ca.gov/dics/epa/epamenu/htm.
9. Gianessi, L.P. and J.E. Anderson. 1995. Pesticide use in U.S. crop protection. National summary report. Washington, D.C.: National Center for Food and Agricultural Policy.
10. Webb, S.R., and J.C. Hall. 1995. Auxinic herbicide-resistant and -susceptible wild mustard (Sinapis arvensis L.) biotypes: Effect of auxinic herbicides on seedling growth and auxin-binding activity. *Pest. Biochem. Physiol.* 52: 137-148.
11. Mullison, W.R. 1985. A toxicological and environmental review of picloram. *Proc. Western Soc. Weed Sci.* 38:27.
12. Gillespie, G.R. 1990. Picloram and clopyralid. *In* W.W. Donald (ed.) *Systems of weed control in wheat in North America.* Monograph Series of the Weed Science Soc. of America. Champaign IL: Weed Science Soc. of America.
13. Hayes, J.R., L.W. Condie, and J.F. Borzelleca. 1986. Acute 14-day repeated dosing, and 90-day subchronic toxicity studies of potassium picloram. *Fund. Appl. Toxicol.* 7:464-470.
14. Chaturvedi, A.K. 1993. Biochemical and toxicological studies on the mixtures of three commonly-used herbicides in mice. *Arch. Environ. Contam. Toxicol.* 24:449-454.
15. Calif. Sept. of Food and Agriculture. Medical Toxicology Branch. 1988. Summary of toxicology data: Picloram. Sacramento, CA, Mar. 7.
16. U.S. EPA. Office of Drinking Water. 1988. Picloram health advisory. Washington DC, Aug.
17. Blakley, P.M., J.S. Kim, and G.D. Firneisz. 1989. Effects of gestational exposure to Tordon 202c on fetal growth and development in CD-1 mice. *J. Toxicol. Environ. Health* 28:309-316.
18. Blakley, P.M., J.S. Kim, and G.D. Firneisz. 1989. Effects of parental subacute exposure to Tordon 202c on fetal growth and development in CD-1 mice. *Teratol.* 39:237-241.
19. Blakley, P.M., J.S. Kim, and G.D. Firneisz. 1989. Effects of preconceptual and gestational exposure to Tordon 202c on fetal growth and development in CD-1 mice. *Teratol.* 39:547-553.
20. U.S. EPA. 1997. Office of Pesticide Programs. Office of Pesticide Programs list of chemicals evaluated for carcinogenic potential. Memo from W.L. Burnam, Health Effects Division, to Health Effects Division Branch Chiefs, et al. Washington, D.C., Feb. 19.
21. U.S. Dept. of Health and Human Services. Public Health Service. Agency for Toxic Substances and Disease Registry. 1996. Toxicological profile for hexachlorobenzene, Aug.
22. National Toxicology Program. 1978. Bioassay of picloram for possible carcinogenicity (CAS No. 1918-02-1). TR-23. http://ntp-server.niehs.nih.gov/htdocs/LT-studies/TR023.html
23. International Agency for Research on Cancer. 1991. Picloram (Group 3) http://193.51.164.11/htdocs/Monographs/Vol53/15-Picloram.html. Updated Nov., 1997.
24. New Jersey Dept. of Health and Senior Services. 1996. Hazardous substance fact sheet: Ethylene glycol. Trenton, NJ, Jan.
25. Acros Organics. 1996. Material safety data sheet. Triisopropanolamine. Fairlawn, NJ, Oct. 3.
26. New Jersey Dept. of Health and Senior Services. 1997. Hazardous substance fact sheet: Isopropyl alcohol. Trenton, NJ, Jan.
27. Jackson, J.B. 1966. Toxicologic studies on a new herbicide in sheep and cattle. *Am. J. Vet. Res.* 27(118):821-824.
28. Newell, K.W., A.D. Ross, and R.M Renner. 1984. Phenoxy and picolinic acid herbicides and small-intestinal adenocarcinoma in sheep. *The Lancet* 2(8415):1301-1304.
29. Woodward, D.F. 1982. Acute toxicity of mixtures of range management herbicides to cutthroat trout. *J. Range Manage.* 35(40:539-540.
30. Mayes, M.A., D.L. Hopkins, and D.C. Dill. 1987. Toxicity of picloram (4-amino-3,5,6-trichloropicolinic acid) to life stages of the rainbow trout. *Bull. Environ. Contam. Toxicol.* 38:653-600.
31. Woodward, D.F. 1976. Toxicity of the herbicides dinoseb and picloram to cutthroat (*Salmo clarki*) and lake trout (*Salvelinus namaycush*). *J. Fish. Res. Board Can.* 33:1671-1676.
32. D.F. Woodward. 1979. Assessing the hazard of picloram to cutthroat trout. *J. Range Manage.* 32(3):230-232.
33. U.S. EPA. Environmental Research Laboratory. 1979. Effects of selected herbicides on smolting of coho salmon. Corvallis OR. Pp. 90-92.
34. U.S. EPA. Office of Pesticide Programs. Ecological Effects Branch. Undated. Ecological Effects Branch science chapter for the reregistration eligibility document (RED) for picloram. Washington, D.C.
35. *Hagen v. Dow Chemical Co.* 1993. 261 Mont. 287, 863 P.2d 413.
36. Keys, B. 1992. Goin' fishin with Tordon 22K: Roadside spraying leaves 15,000 pounds of trout dead. *JPR* 12(2):18-19.
37. Gardner, G.R. et al. 1991. Germinomas and teratoid siphon anomalies in softshell clams, *Mya arenaria*, environmentally exposed to herbicides. *Environ. Health Persp.* 90:43-51.
38. Van Beneden, R.J. 1993. Implications for the presence of transforming genes in gonadal tumors in two bivalve mollusk species. *Cancer Res.* 53:2976-2979.
39. Sheets, T.J. and B.J. Harrell. 1986. Effects of low levels of soil-applied picloram on flue-cured tobacco. Tech. Bull. 280. Raleigh, NC: North Carolina State University. North Carolina Agricultural Research Service.
40. Jacoby, P.W., C.H. Meadors, and L.E. Clark. 1990. Effects of triclopyr, clopyralid, and picloram on growth and production of cotton. *J. Prod. Agric.* 3(3):297-299.
41. Harvey, G.R. and J.D. Mann. 1968. Picloram in Vietnam. *Scientist and Citizen* 10:165-171.
42. Kerns, J. 1988. Who is liable for $250,000 of potatoes? *JPR* 7(4):10-13.
43. Tomkins, D.J. and W.F. Grant. 1976. Monitoring natural vegetation for herbicide-induced chromosome aberrations. *Mut. Res.* 36:73-84.
44. Tu, C.M. 1994. Effects of herbicides and fumigants on microbial activities in soil. *Bull. Environ. Contam. Toxicol.* 53:12-17.
45. Forsyth, D.J., P.A. Martin, and G.G. Shaw. 1997. Effects of herbicides on two submerged aquatic macrophytes, *Potamogeton pectinatus* L. and *Myriophyllum sibiricuym* Komarov, in a prairie wetlands. *Environ. Pollut.* 95:259-268.
46. Cullimore, D.R. 1975. The *in vitro* sensitivity of some species of Chlorophyceae to a selected range of herbicides. *Weed Res.* 15:401-406.
47. Fuerst, E.P. et al. 1996. Physiological characterization of picloram resistance in yellow starthistle. *Pest. Biochem. Physiol.* 56:149-161.
48. Hall, J.C., S.R. Webb, and S. Deshpande. 1996. An overview of auxinic herbicide resistance: Wild mustard (*Sinapis arvensis* L.) as a case study. Chap 4. *In* Brown, T.M. (ed.) *Molecular genetics and evolution of pesticide resistance.* ACS Symposium Series 645. Washington, D.C. American Chemical Society.
49. U.S. EPA. Office of Pesticide Programs. Environmental Fate and Ground Water Branch. Undated. Environmental Fate and Ground Water Branch review of picloram. Washington, D.C.
50. Cryer, S.A. et al. 1992. Picloram movement and dissipation in a northern rangeland environment. *Western Soc. Weed Sci.* 45:120-121.
51. Johnsen, T.N. 1980. Picloram in water and soil from a semiarid pinyon-juniper watershed. *J. Environ. Qual.* 9(4):601-605.
52. Michael, J.L., D.G. Neary, and J.M Wells. 1989. Picloram movement in soil solution and streamflow from a coastal plain forest. *J. Environ. Qual.* 18:89-95.
53. Hickman, M.V., C.G. Messersmith, and R.G. Lym. 1989. Picloram release from leafy spurge (*Euphorbia esula*) roots in the field. *Weed Sci.* 37:167-174.
54. Webb, W.L. and M. Newton. 1972. Release of picloram from roots. *Weed Res.* 12:391-394.
55. Lavy, T.L. et al. 1996. Long-term *in situ* leaching and degradation of six herbicides aged in subsoils. *J. Environ. Qual.* 25:1268-1279.
56. U.S. Dept. of the Interior. U.S. Geological Survey. National Water-Quality Assessment Program. 1994. Assessment of selected constituents in surface water of the upper Snake River Basin, Idaho and western Wyoming, water years 1975-1989. Water-Resources Investigations Report 93-4229. Boise, ID.
57. Mehnert, E. et al. 1995. The occurrence of agricultural chemicals in Illinois' rural private wells: Results from the pilot study. *Ground Water Monitor. Rev.* 15(1):142-149.
58. U.S. EPA. Prevention, Pesticides and Toxic Substances. 1992. Pesticides in groundwater database. A compilation of monitoring studies: 1971-1991. National summary. Washington, D.C.
59. Letter from M.L. McGlothlin, Oregon Dept. of Environmental Quality, to Neva Hassanein, NCAP. May 20, 1992.
60. U.S. Dept. of the Interior. U.S. Geological Survey. National Water-Quality Assessment Program. 1996. Pesticides in public supply wells of Washington State. Factsheet FS-122-96. Tacoma, WA.
61. Letter from Mary Toohey, Washington Dept. of Agriculture, to Gil Zemansky, Friends of the Earth. Dec. 13, 1979.
62. Frank, R. et al. 1987. Investigations of pesticide contaminations in rural wells, 1979-1984, Ontario, Canada. *Arch. Environ. Contam. Toxicol.* 16:9-22.
63. Smith, A.E. et al. 1988. Persistence and movement of picloram in a northern Saskatchewan watershed. *J. Environ. Qual.* 17(2):262-268.
64. Kohl, R.A., C.G. Carlson, and S.G. Wangemann. 1994. Herbicide leaching potential through road ditches in thin soils over an outwash aquifer. *Appl. Eng. Agricul.* 10(4):497-503.
65. Dennis, D.S. 1977. Accumulation and persistence of picloram (Tordon 10K) in surface water and bottom sediments in West Virginia. *Arch. Environ. Contam. Toxicol.* 6:421-433.
66. Lym, R.G. and C.G. Messersmith. 1988. Survey for picloram in North Dakota groundwater. *Weed Technol.* 2:217-222.
67. Larson, S.J., P.D. Capel, and M.S. Majewski. 1997. *Pesticides in surface waters: Distribution, trends, and governing factors.* Chelsea, MI: Ann Arbor Press.