**PICLORAM (TORDON, GRAZON) (EU: Galera; authorized on rapeseed in Eastern Europe & UK only)**

n.b.: the Anglo meanings of numbers’ commas & periods are the reverse of the Continent’s use.

FEIS Claim: "**Carcinogenic**: Unknown"

DAR Claim: NOAEL 60 mg/kg bw d- (pre-cancerous signs); cancer around 1,000 mg/kd bw d-)

What the Literature Says: The National Toxicology Program and World Health Organization (WHO’s Int’l Agency for Research on Cancer, IARC) cancer assays are both regarded as the gold standard of cancer tests. Both of these independent tests found liver tumors in picloram exposed test animals, with IARC additionally finding thyroid tumors; but doses were very high—thousands of mg/kg bw d-.[[1]](#footnote-1) NTP concluded carcinogenicity evidence was equivocal; IARC that picloram shows limited evidence of carcinogenicity and is currently unclassifiable. But IARC reviewed a published LOAEL (i.e. no lower dose studied) for cancers in rats at just 20 mg/kg bw d-, with a clear dose/response relation up through the highest dose of 200 mg/kg.[[2]](#footnote-2)

Picloram’s manufacture results in **hexachlorobenzene (HCB) contamination**; HCB is a probable human carcinogen according to EPA’s Office of Pesticide Program’s 1997 list of chemicals evaluated for carcinogenic potential, which estimates that HCB in picloram alone accounts for 70% of EPA’s allowable risk for HCB exposure.[[3]](#footnote-3) The exposure to HCB of ground applicators of picloram to exceed EPA’s acceptable cancer risk level by ten-fold.[[4]](#footnote-4) But picloram’s HCB contamination today may be a bit lower.

The DAR concludes that HCB exposure from picloram poses no real risk of cancer, based first on no or benign tumors only in experiments using picloram; and second on tests directly on HCB finding tumors only at 1.5 mg/kg bw d-, concluding that doses of HCB from use picloram are some thousand-fold lower.

Yet USEPA’s NOAEL for HCB is 0.08 mg/kg bw d- (for necrosis & chromosome damage), i.e. 19 times more potent. HCB at 0.02 ppb in drinking water is calculated to cause cancer in one-in-a million animals. In sum, picloram’s carciingenic HCB risk is greater than recognized. As it is also mutagenic (see below), the DAR, by assuming it was not, failed to evaluate this risk in a no-threshold mode, where the cancer risks may exceed the acceptable one-in-a-million. And If the DAR had also considered cumulative HCB exposure, it may have concluded this exposure source needs limiting.

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FEIS Claim: "**Teratogenic**: No Effects"

DAR Claim: “None.”

What the Literature Says: Picloram caused umbilical hernias at all dose levels and multiple skeletal malformations at both high and low doses,[[5]](#footnote-5) while male rats suffered atrophied testicles.[[6]](#footnote-6) Picloram plus 2,4-D (‘Tordon 202c’ brand) is a teratogen at 0.21% and above (no lower dose tested) in drinking water, in utero and even preconception--even to the male alone.[[7]](#footnote-7)

FEIS Claim: "**Reproductive**: No Effects"

DAR Claim: NOAEL 200 mg/kg bw d-

DAR Claim (Developmental) 30 mg/kg bw d-, (liver growth & hyperplasia)--the lowest NOAEL, and so is the basis for picloram’s ADI of 0.3 mg/kg bw d- ADI.

What the Literature Says: A re-review of National Cancer Institute testicular slides of picloram exposed rats and mice determined that many of the animals had testicular atrophy,[[8]](#footnote-8) after initially finding no atrophy; a result that the manufacturer Dow disputed. Dow did find increased miscarriages at picloram the higher test dose/s,[[9]](#footnote-9) and the State of California found increased embryo loss for the potassium salt formulation of the picloram molecule.[[10]](#footnote-10) The above picloram + 2,4-D birth defect doses also caused pregnancy losses. These same two a.i. sold as the Tordon75D formulation are severely toxic to test animal testicles.[[11]](#footnote-11)

**HCB** allowed at up to 200 ppm in picloram persistent and already associated with several human reproductive disorders; was shown to significantly speed sexual maturity of the prostate in male mice at 20 mg/kg bw d-, yet it significantly retarded this development at a dose just two to greater than 20 times higher! In vitro, this strange response was confirmed as just 0.5 - 5 nM HCB (a realistic human dose) stimulated action via the androgen hormone receptor (a prostate cancer risk) while 10 uM and higher doses repressed it.[[12]](#footnote-12)

FEIS Claim: "**Mutagenic**: Unlikely"

DAR claim: “No evidence” for both picloram and its contaminant HCB

What the Literature Says: The National Toxicology Program found that chromosome aberrations and sister chromatid exchanges (SCEs) increased in frequency in hamster ovary cells exposed to picloram.[[13]](#footnote-13) Picloram twice again tested positive for mutagenicity in tests.[[14]](#footnote-14) Picloram’s contaminant **HCB** has tested mutagenic,[[15]](#footnote-15) contrary to Dow & DAR’s claim. NTP and others have found chromosome damge by picloram.

It is worth noting that **EPA’s official non-cancer safe dose estimate (RfD) for picloram is 0.07 mg/kg of body weight per day, fully cited in EPA’s Integrated Risk Information System (IRIS) database**. But the main Q-RA that the USFS is relying on uses a picloram RfD almost 3 times higher (less safe), 0.2 mg/kg b.w./day (which happens to be the same as Dow’s RfD, the manufacturer of picloram. Nevertheless, even at that “safe” dosage there are a few modeled exposures in that Q-RA (which models exposures of pesticide applicators, the general human population, aquatic and terrestrial organism) that exceed the more lenient RfD. How many more would exceed it if they had used the stricter RfD?

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**PICLORAM**: a CASE STUDY of FAILURE of FIFRA's “NO UNREASONABLE RISK” MANDATE

Aside from this FEIS’ many claims of herbicide safety due to registration and of the unquantified ability of mitigation to take care of any consequences that do occur, this FEIS claims that the impacts of herbicides on non-target vegetation will be transient[[16]](#footnote-16) and outweighed by the benefit to native vegetation from herbicide use[[17]](#footnote-17) (which ignores other methods of control and ignores that herbicide use is not an effective control in the long run, as proved above). The authorities to prove these claims are not given.

Dr. Peter Rice at the U. of Montana, Missoula MT, has accepted at least $24,000 dollars from Dow,[[18]](#footnote-18) picloram’s manufacturer, to do research on picloram’s non-target vegetation toxicity. Picloram is the most widely used herbicide on western public lands, including those of the USFS.[[19]](#footnote-19) It was recommended to be banned by EPA’s experts on the environmental behavior of pesticides, due precisely to its persistence and toxicity in water and its non-target plant toxicity.[[20]](#footnote-20) So, if glyphosate is even less selective than picloram, as this section of the FEIS says, what’s the impact of glyphosate on non-target environments? Now we show just how non-selective picloram is.

There seems to be no similar exaggeration by the DAR that picloram is selective. But it estimates that at 1 meter form the application spray non-target plant exposure is 7.9 times less than its toxic level to plants, a bit safer than the allowable 5X.

The chlorinated pyridine family of herbicides are to be heavily relied on in this FEIS (picloram, triclopyr and clopyralid). The FEIS calls, without citation, clopyralid the most selective herbicide, i.e. the least harmful to non-target vegetation) are persistent (especially in water) and mobile in the environment; and kill non-target plants of intrinsic and economic value.[[21]](#footnote-21) There are many instances of them contaminating groundwater and killing native plants and crops. Examples are that the state of Washington recently banned clopyralid after several incidents of it poisoning plants and crops even after surviving the microbial and heat decomposition of compost; after which Dow was forced to announce the voluntary withdrawal of clopyralid’s registration for use on residential lawns nation-wide.[[22]](#footnote-22) Picloram was recently detected in the *drinking* water well of a St Charles, IL school, at over twice the allowable level.[[23]](#footnote-23) Locally, a recent large native vegetation kill occurred at the BNF’s ‘Willoughby 40’ Recreation Area after a picloram + 2,4-D application.[[24]](#footnote-24) When considering whether to ban picloram (during its re-registration), EPA calculated that it exceeds the EPA ‘substantial hazard’ level to non-target plants by up to 13,000(!) times, depending on the application method.[[25]](#footnote-25) the Chief of the Ecological Effects Branch of pesticide registration recommended against the re-registration (i.e., a ban) of picloram:

"This conclusion is based on the extreme exceedance of the acute levels of concern for non-endangered and endangered terrestrial plants."[[26]](#footnote-26)

Critically, a recent local study found that a common picloram formulation, applied according to the label, kills native plant germination even two years after its application (using the soil half-life of picloram to derive the 2nd year’s concentration, as there was an external time limit to the experiment),[[27]](#footnote-27) confirming what the picloram label (and much of the literature the Forest cites, such as the Rice paper) agree is a major risk of picloram (non-target plant risk).

Thus, the consequences of herbicides on native plants have not been independently demonstrated in this FEIS. Because clopyralid and triclopyr are in the same chemical family as picloram and have similar persistence and mobility in the environment;[[28]](#footnote-28) and given the multiple examples of clopyralid & triclopyr’s effects on non-target vegetation, *even after being on other plants and composted*; we are highly skeptical of the FEIS’ un-cited claim that clopyralid is the most selective herbicide currently planned for use. We expect all of picloram’s ecological dangers that led EPA scientists to recommend it be banned would be somewhat similar for the other members of this chemical family.

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The other driver of the EPA scientists’ desire to ban picloram is its persistence and mobility:

The DAR predicts less than 0.1 ug/L picloram will reach 1 m deep into soil after a rapeseed season’s applications, implying that there is no risk to aquatic organisms from runoff; and says its degradation in water is moderately slow with a half-life of 300 days. The DAR then states that 0.55 mg/L picloram (fish, chronic NOEC) is the lowest dose toxic to any aquatic species.

In the EPA’s re-registration opinion, later politically over-ruled, these scientists concluded:

"...because its use would pose unreasonable adverse effects to the environment. Because of picloram's mobility in soil types and its persistence under normal ambient conditions, no practical use restriction can prevent it from contaminating the environment surrounding the target site...The use pattern of picloram is highly specialized, but it is almost certain to eventually reach ground water in areas where it persists in the overlying soil. In submitted terrestrial field and forestry studies, picloram exhibited calculated half-lives of up to 278 days and was detected up to the limits of sampling depth (up to 1.8 m)...Picloram has a high degree of phytotoxicity. ... [Picloram is] resistant to biotic and abiotic (e.g., u.v.) degradation processes. ...In some soils, it is nearly recalcitrant to all degradation processes.”[[29]](#footnote-29)

Thus picloram contaminates water easily--e.g. it was in 420 of 744 surface water samples in EPA’s national STORET drinking water database, found at up to 30 ppb.[[30]](#footnote-30) A field study found that 6% of applied picloram left an 8-acre treated site contaminated over a month with several rainstorms. Measured at the entry point into the stream, it diluted to 5 ppb a full 5.4 km downstream.[[31]](#footnote-31) Given that eight times that level (40 ppb or 0.04 mg/L) kills and decimates trout fry,[[32]](#footnote-32) what were the aquatic effects of this washed-off picloram in the several km. upstream? That fish acute toxicity level is 13 times lower than the DAR’s alleged toxic dose for chronic exposures (also the alleged most sensitive concentration for aquatic species)!

In a study of picloram's persistence and mobility, in Western Montana, found that picloram applied at recommended rates persisted and migrated downwards towards groundwater as far and as long as was measured (over 10% of application detected at one meter after 90 days (and increasing at this depth as measurement ceased); 10% detected in top 12.5 cm of soil after 445 days at a second site),[[33]](#footnote-33) a study that EPA scientists cited in their recommendation that picloram be banned. Finally, two week-apart applications of picloram a1/4 mile upstream of a fish hatchery killed 15,000 pounds of trout after a rainstorm flushed it into the water.[[34]](#footnote-34)

**HCB contaminant**: It has been shown that wildlife (just as EPA calculated for human picloram applicators) are at great risk of cancer or other disease from HCB. Predators of ungulates eating only picloram-sprayed forage were, in a local Environmental Assessment (EA), calculated to ingest 0.021 to 0.034 mg/kg per daily meal of HCB--up to 43 times the HCB non-cancer ‘safe dose’ (RfD) of 0.0008 mg/kg/d. The ungulates were claimed to ingest less than the HCB safe dose, without any quantification; and the Finding of No Significant Impact (FONSI) to avoid an EIS was finalized![[35]](#footnote-35) Exposure to HCB of ground applicators of picloram exceeds EPA’s acceptable cancer risk level by ten-fold (HCB causes cancer in test animals at very low doses: 0.02 ppb in drinking water is calculated to cause cancer in one-in-a million animals). These applicator’s intermediate-term HCB exposure also exceeds the ATSDR’s non-cancer safe dose level.[[36]](#footnote-36) The DAR states that applicators are exposed to “acceptable” levels of picloram even without clothing or respirator protection, leaving this HCB risk unanalyzed.

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Picloram’s new label (re-registration was granted in 1995) contains many examples of ambiguous use directions, of the type that recently caused EPA to begin to crack-down on such un-enforceable label directions (see the above criticisms). “Application *should* be avoided [at winds] below 2 mph [to minimize direction shifts and temperature inversions]...and above 10 mph.” This label advisory, in place of a clear use direction, means there is no need to worry about how to measure wind speed, or if it is possible to keep wind speed and direction constant. Picloram’s label also, inter alia, “advises” not to apply to residential or commercial lawns; “advises” drift be avoided; and says to apply at rates “suggested” in the approved uses section of the label (though, still more ambiguously, application rates are elsewhere in the label mandated). These unenforceable label use advisories were issued by EPA in the face the original recommendation of its scientists to ban picloram, but for exactly the same reasons.

Instead of a ban, EPA over-rode its experts on environmental fate and ecological effects, and re-registered picloram. EPA restricted its manner of application somewhat (both with mandatory and ambiguous advisory label language), and called for several studies on these questions of picloram’s risks.[[37]](#footnote-37) As explained above in the general critique of FIFRA , calling for studies while registering a pesticide for use appears to be an example of an illegal registration: if more studies on these critical effects of picloram use are required, how on earth can EPA have determined that there is “no unreasonable risk”? The only other major factor in this registration determination is that of Dow’s economic benefit.

**In sum**, picloram’s persistence and mobility allow it to encounter all the life forms of an ecosystem, and it has been shown to be toxic to many of these organisms at the levels it is applied. It is well known that weeds are developing resistance to it. As EPA’s scientists wanted, it should be banned.

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2. Stott, WT et al. (1990) Chronic toxicity and oncogenicity of picloram in Fischer 344 rats.! Toxicol. environ. Health, 30,91-104 [↑](#footnote-ref-2)
3. EPA/OPP 1996 Picloram RED. [↑](#footnote-ref-3)
4. EPA/OPP 1996 Picloram RED. [↑](#footnote-ref-4)
5. California Dpt. of Food & Agriculture Medical Toxicology Branch 1988 ‘Summary of Toxicological Data, Picloram’ Sacramento CA. [↑](#footnote-ref-5)
6. EPA/Office of Drinking Water (ODW) 1988 ‘Picloram Health Advisory’ Wash. DC. [↑](#footnote-ref-6)
7. Blakley P et al. 1989 (3 papers): Teratology:39-237-41 **and** 39:547-53; **and** J. Tox. & Env. Health:28:309-16. [↑](#footnote-ref-7)
8. M. Reuber 1981 ‘Carcinogenicity of Picloram’ J. Toxicol. & Env. Health:7:2:207-222. [↑](#footnote-ref-8)
9. EPA 1995 (Picloram RED). [↑](#footnote-ref-9)
10. Calif. DF&A 1988. [↑](#footnote-ref-10)
11. Oakes et al. 2002 (a series of papers). [↑](#footnote-ref-11)
12. Jody Ralph et al April 2003 ‘Disruption of Androgen Regulation in the Prostate by the Env. Contaminant Hexachlorobenzene’ Env. Health Perspectives 111:4:461-466.. [↑](#footnote-ref-12)
13. Calif. DF&A 1988. [↑](#footnote-ref-13)
14. Muhammed et al. 1993 Mutat. Res.:426:2:193-199; and Verikat et al. 1995 Environ. Mol. Mutagen.:25:1:67-76. [↑](#footnote-ref-14)
15. Canonero R, et al. 1997 Jan ‘Testing of p-dichlorobenzene and hexachlorobenzene for their ability to induce DNA damage and micronucleus formation in primary cultures of rat and human hepatocytes.’ Mutagenesis:12(1):35-9. [↑](#footnote-ref-15)
16. FEIS p. 4-5. [↑](#footnote-ref-16)
17. FEIS p. 4-5. [↑](#footnote-ref-17)
18. U. Montana/Integrated Plant Management Committee 1998 ‘Mt. Sentinel Vegetation Management Final Plan’ Sec. 4.17 (pg. 17). Will Snodgrass (Chemical Injury Information Network, Missoula MT) exposed this conflict of interests. [↑](#footnote-ref-18)
19. Cooperative Extension Services of MT, UT & WY ‘1999-2000 Weed Management Handbook’. [↑](#footnote-ref-19)
20. EPA/OPP 1995 ‘Re-Registration Eligibility Document (RED) for picloram’ Wash. DC. [↑](#footnote-ref-20)
21. EPA/OPP 1996 ‘Picloram RED Facts’, Wash. DC (the Re-registration Eligibility Document (RED) factsheet). [↑](#footnote-ref-21)
22. J. Pesticide Reform, Fall 2002, p.5. **Also** David Bezdicek, 6/04/2001 ‘New Herbicides Found in WSU Compost’ Washington State U Compost Facility home page, index.htm --many other examples exist. [↑](#footnote-ref-22)
23. NCAMP/Beyond Pesticides Technical Rpt., May 2002. [↑](#footnote-ref-23)
24. Ravalli Republic, 6 June 2001, p. 1. [↑](#footnote-ref-24)
25. EPA/OPP 1995 (Picloram RED). [↑](#footnote-ref-25)
26. EPA/OPP, Special Review and Registration Division, Ecological Effects Branch undated ‘Review of Picloram’ (prepared for the 1995 re-registation of picloram). [↑](#footnote-ref-26)
27. Amanda Tripp 2003 ‘The Use of Blanketflower (G. aristata) As a Bioassay for Residual Tordon 22K’, presented of MT Academy of Sciences Annual Meeting, Junior Section, 26 Apr. 2003, U. Montana Missoula MT (Big Sky HS student). [↑](#footnote-ref-27)
28. EPA/OPP 1998 ‘Triclopyr RED Factsheet’;.EPA 16 May 1997 ‘Clopyralid; Pesticide Tolerance...’ Fed. Reg. 62:26,949-26,954. [↑](#footnote-ref-28)
29. EPA/OPP, Special Review and Registration Division, Env. Fate & GroundWater Branch (EF&GWB) undated ‘Review of Picloram’ (prepared for the 1995 re-registation of picloram). [↑](#footnote-ref-29)
30. EPA/OPP 1996 (Picloram R.E.D. Factsheet). [↑](#footnote-ref-30)
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32. D. Woodward 1976 ‘Toxicity of the Herbicides Dinoseb & Picloram...’ J. Fish. Res. Board Can. 33:1671-16. [↑](#footnote-ref-32)
33. Vicky Watson et al. 1989 ‘Env. Fate of Picloram Used for Roadside Control’ J. of Env. Quality 18:198-205. [↑](#footnote-ref-33)
34. B. Keyes 1992 'Going Fishin' With Tordon 22K' J Pesticide Reform:12:2:18-19. [↑](#footnote-ref-34)
35. U. of Montana/Integrated Plant Management Committee May 1998 ‘Mt. Sentinel Vegetation Management Final Plan’. See response to comment #1.8 on p. 5. [↑](#footnote-ref-35)
36. EPA/OPP 1996 Picloram RED. [↑](#footnote-ref-36)
37. EPA/OPP 1996 (Picloram RED Factsheet). [↑](#footnote-ref-37)