7. *Metabolite toxicology*. The residue of concern for tolerance setting purposes is the parent material (diclosulam). Thus, there is no need to address metabolite toxicity.

C. Aggregate Exposure

1. *Dietary exposure—Food*. For Purposes of assessing the potential dietary exposure from use of diclosulam on soybeans and peanuts, a conservative estimate of aggregate exposure is determined by Theoretical Maximum Residue Contribution (TMRC) assuming that 100% of the soybeans and peanuts have a residue of diclosulam at the proposed tolerance level of 0.02 ppm. This results in an extremely conservative estimate of exposure for diclosulam, because no residues are expected in these commodities at the proposed maximum label rate. The potential dietary exposure is obtained by multiplying the tolerance residue level on soybeans and peanuts (0.02 ppm) by the consumption data which estimates the amount of soybean and peanut products consumed by various population subgroups. The maximum potential average daily dose (ADD) of diclosulam values determined for various populations are clearly significant overestimates compared with actual exposure. When ADDs are compared to the Reference Dose (RfD), which uses the lowest NOAEL of 5 mg/kg/day from the 2-year rat chronic toxicity study and an uncertainty factor of 100, the ADD for all U.S. consumers including the highest exposed group, non-nursing infants under 1-year old, would theoretically be exposed to about 0.1% of the RfD.

2. *Drinking water*. Another potential source of dietary exposure are residues in drinking water. Based upon the available field dissipation and field run off studies conducted with diclosulam there is little potential for exposure to diclosulam in drinking water to cause any human health concern.

D. Cumulative Effects

There is no reliable information to indicate that diclosulam has a common mechanism of toxicity with any other chemical compound or that potential toxic effects of diclosulam would be cumulative with those of any other pesticide chemical. Thus Dow AgroSciences believes it is appropriate to consider only the potential risks of diclosulam in its exposure assessment.

E. Safety Determination

1. *U.S. population*. Using the conservative exposure assumptions described above, and based on the completeness and reliability of the toxicity data, Dow AgroSciences has concluded that aggregate exposure to diclosulam potentially can utilize about 0.1% of the RfD for non-nursing infants under 1-year old, theoretically the most exposed population. EPA generally has no concern for exposures below 100% of the RfD because the RfD represents the level at or below which daily aggregate dietary exposure over a lifetime will not pose appreciable risks to human health. Therefore, Dow AgroSciences concludes that there is a reasonable certainty that no harm will result from aggregate exposure to diclosulam residues in on soybeans and peanuts and its processed products.

The complete toxicology profile for diclosulam shows no evidence of physiological effects characteristic of the disruption of the hormone estrogen. Based upon this observation, diclosulam does not meet the criteria for an estrogenic compound.

2. *Infants and children*. In assessing the potential for additional sensitivity of infants and children to residues of diclosulam, data from developmental toxicity studies in rats and rabbits and a multigeneration reproduction study in the rat are considered. The developmental toxicity studies are designed to evaluate adverse effects on the developing organism resulting from pesticide exposure during prenatal development to one or both parents. Reproduction studies provide information relating to effects from exposure to the pesticide on the reproductive capability and potential systemic toxicity of mating animals and on various parameters associated with the well-being of offspring.

FFDCA section 408 provides that EPA may apply an additional safety factor for infants and children in the case of threshold effects to account for pre- and post-natal toxicity and the completeness of the data base. Based on the current toxicological data requirements, the data base for diclosulam relative to pre- and post-natal effects for children is complete. Further, for diclosulam, the NOAEL in the chronic feeding study which was used to calculate the RfD (5 mg/kg/day) is already lower than the NOAELs from the developmental studies in rats and rabbits by a factor of more than 200-fold.

Concerning the reproduction study in rats, there were no effects on reproduction or fetal development, even at a dose over 100 times the NOAEL used to establish the RfD. Therefore, Dow AgroSciences concludes that an additional uncertainty factor is not needed and that the RfD at 0.05 mg/kg/day is appropriate for assessing risk to infants and children.

Using the conservative exposure assumptions previously described, the percent RfD utilized by the aggregate (diet, and drinking water) exposure to residues of diclosulam on soybeans and peanuts is 0.000051 mg/kg/day for non-nursing infants under 1-year old, theoretically the most exposed population subgroup. Thus, based on the completeness and reliability of the toxicity data and the conservative exposure assessment, Dow AgroSciences concludes that there is a reasonable certainty that no harm will result to infants and children from aggregate exposure to diclosulam on soybeans and peanuts.

F. International Tolerances

There are no Codex maximum residue levels established for residues of diclosulam on soybeans, peanuts or any other food or feed crop.

[FR Doc. 98–31066 Filed 11–19–98; 8:45 am]
BILLING CODE 6560–50–F

ENVIRONMENTAL PROTECTION AGENCY

[PF–840; FRL–6039–6]

Dow AgroSciences LLC; Pesticide Tolerance Petition Filing

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice.

SUMMARY: This notice announces the initial filing of a pesticide petition proposing the establishment of regulations for residues of a certain pesticide chemical in or on various food commodities.

DATES: Comments, identified by the docket control number PF–840, must be received on or before December 21, 1998.

ADDRESSES: By mail submit written comments to: Information and Records Integrity Branch, Public Information and Services Divison (7502C), Office of Pesticides Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460. In person bring comments to: Rm. 119, CM #2, 1921 Jefferson Davis Highway, Arlington, VA.

Comments and data may also be submitted electronically by following the instructions under ''SUPPLEMENTARY INFORMATION.'' No confidential business information should be submitted through e-mail.

Information submitted as a comment concerning this document may be claimed confidential by marking any part or all of that information as ''Confidential Business Information'' (CBI). CBI should not be submitted

through e-mail. Information marked as CBI will not be disclosed except in accordance with procedures set forth in 40 CFR part 2. A copy of the comment that does not contain CBI must be submitted for inclusion in the public record. Information not marked confidential may be disclosed publicly by EPA without prior notice. All written comments will be available for public inspection in Rm. 119 at the address given above, from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays.

FOR FURTHER INFORMATION CONTACT: James A. Tompkins, Herbicide Branch, Registration Division (7505C), Office of Pesticide Programs, Environmental Protection Agency, 401 M St., SW, Washington, DC 20460. Office location, telephone number, and e-mail address: Rm. 239, Crystal Mall #2, 1921 Jefferson Davis Highway, Arlington, VA 22202, (703) 305–5697; e-mail: tompkins.jim@epamail.epa.gov.

SUPPLEMENTARY INFORMATION: EPA has received a pesticide petition as follows proposing the establishment and/or amendment of regulations for residues of certain pesticide chemical in or on various food commodities under section 408 of the Federal Food, Drug, and Comestic Act (FFDCA), 21 U.S.C. 346a. EPA has determined that this petition contains data or information regarding the elements set forth in section 408(d)(2); however, EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data supports granting of the petition. Additional data may be needed before EPA rules on the petition.

The official record for this notice of filing, as well as the public version, has been established for this notice of filing under docket control number [PF–840] (including comments and data submitted electronically as described below). A public version of this record, including printed, paper versions of electronic comments, which does not include any information claimed as CBI, is available for inspection from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays. The official record is located at the address in ''ADDRESSES'' at the beginning of this document.

Electronic comments can be sent directly to EPA at:

opp-docket@epamail.epa.gov

Electronic comments must be submitted as an ASCII file avoiding the use of special characters and any form of encryption. Comment and data will also be accepted on disks in Wordperfect 5.1/6.1 file format or ASCII file format. All comments and data in electronic form must be identified by the docket control number (PF–840) and appropriate petition number. Electronic comments on this notice may be filed online at many Federal Depository Libraries.

List of Subjects

Environmental protection, Agricultural commodities, Food additives, Feed additives, Pesticides and pests, Reporting and recordkeeping requirements.

Dated: October 22, 1998.

James Jones,

Director, Registration Division, Office of Pesticide Programs.

Summary of Petition

The petitioner summary of the pesticide petition is printed below as required by section 408(d)(3) of the FFDCA. The summary of the petition was prepared by the petitioner and represents the views of the petitioner. EPA is publishing the petition summaries verbatim without editing them in any way. The petition summary announces the availability of a description of the analytical methods available to EPA for the detection and measurement of the pesticide chemical residues or an explanation of why no such method is needed.

1. Dow AgroSciences LLC

PP 4F4412

On May 13, 1997 (62 FR 26305) EPA published a notice that EPA had received pesticide petition (PP 4F4412) from Dow AgroSciences, 9330 Zionsville Road, Indianapolis, IN 46268-1054, proposing pursuant to section 408(d) of the Federal Food, Drug and Cosmetic Act, 21 U.S.C. 346a(d), to amend 40 CFR part 180 by establishing a tolerance for inadvertent residues of the herbicide picloram in or on the raw agricultural commodity grain sorghum grain, forage, and stover at 0.3, 0.2, and 0.5 parts per milliom (ppm), respectively. No comments were received to the initial notice of filing. This notice announces that the petition was amended by also proposing to establish a tolerance for residues of the herbicide picloram in or on the raw agricultural commodity aspirated grain fractions at 4 ppm. The analytical method is Method A and III listed in the Pesticide Analytical Manual (PAM), Vol. II. EPA has determined that the petition contains data or information regarding the elements set forth in section 408(d)(2); however, EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data supports granting of the petition. Additional data may be needed before EPA rules on the petition.

A. Residue Chemistry

1. *Plant metabolism*. The qualitative nature of the residue in plants is understood based on a wheat metabolism study. The residue of concern in wheat forage, straw and grain is conjugated picloram, which is hydrolyzable by acid, base and B-glucosidase. The minor metabolites that were identified in grain and straw were 4-amino-6-hydroxy-3,5-dichloropicolinic acid and 4-amino-2,3,5-trichloropyridine.

2. *Analytical method*. The analytical portions of the magnitude of residue studies were performed at Dow AgroSciences in Midland, MI. The analytical method utilized for the determination of picloram residue levels in the submitted studies was ACR 73.3.S2. There is a practical analytical method for detecting and measuring levels of picloram in or on food with a limit of quantitation that allows monitoring of food with residues at or above the levels set in these tolerances. EPA has provided information on this method to FDA. The method is available to anyone who is interested in pesticide residue enforcement.

3. *Magnitude of residues*.

SUMMARY OF RESIDUES OF PICLORAM (PPM) FOUND IN GRAIN SORGHUM

Matrix	Range
Grain	ND[a]-0.23
Forage	ND-0.17
Fodder	ND-0.44

[a]ND = less than one-half of the validated lower limit of quantitation of 0.05 μg/g in grain and 0.1 μg/g in forage and fodder.

B. Toxicological Profile

1. *Acute toxicity*. Studies for acute toxicity indicate that picloram is classified as category III for acute oral toxicity, category III for acute dermal toxicity, category I/II (depending on whether acid or salts) for acute inhalation toxicity, category IV for skin irritation potential, and category III for eye irritation potential. The potassium salt is classified as a skin sensitizer. In addition, picloram has a low vapor pressure.

Picloram potassium salt has low acute toxicity. The rat oral LD_{50} is 3,536 milligrams/kilogram (mg/kg) or greater for males and females. The rabbit dermal LD_{50} is >2,000 mg/kg and the rat inhalation LC_{50} is >1.63 mg/L air (the highest attainable concentration).

Picloram potassium salt is a positive skin sensitizer in guinea pigs but is not a dermal irritant. Technical picloram potassium salt is a moderate ocular irritant but ocular exposure to the technical material would not normally be expected to occur to infants or children or the general public. End use formulations of picloram have similar low acute toxicity profiles plus low ocular toxicity as well. Therefore based on the available acute toxicity data, picloram does not pose any acute dietary risks.

2. *Genotoxicty*. Picloram acid was evaluated in the Ames test using *Salmonella typhimurium*. Doses ranged up to 5,000 µg/plate, with and without metabolic activation. The test substance did not produce a mutagenic response either in the presence or absence of activation.

Picloram acid was evaluated for gene mutation in mammalian cells (HGPRT/CHO). As evaluated up to toxic levels (1,750 µg/ml without metabolic activation; 4,500 µg/ml with metabolic activation), the compound was found to be negative for inducing forward mutation in Chinese hamster ovary (CHO) cells.

Picloram acid was evaluated for cytogenetic effects on bone marrow cells of rats via intra gastric administration at dosage levels of 0 (vehicle), 20, 200 or 2,000 mg/kg. The test material did not produce cytogenetic effects in the study.

Picloram acid was evaluated for genotoxic potential as administered to primary rat hepatocyte cultures at concentrations of 0 (vehicle), 10, 33.3, 100, 333.3 or 1,000 µg/ml. The test material was negative for unscheduled DNA synthesis (UDS, a measure of DNA damage/repair) treated up to cytotoxic levels of (1,000 µg/ml).

3. *Reproductive and developmental toxicity*. The HED reference dose (RfD) Peer Review Committee concluded that there was no evidence, based on the available data, that picloram and its salts were associated with significant reproductive or developmental toxicity under the testing conditions.

In the following developmental toxicity studies, the dose levels that appear in parenthesis are picloram acid equivalents where the conversion factor employed was 0.86 as applied to doses of potassium salt.

Picloram potassium salt was administered to New Zealand rabbits by oral gavage at dosage levels of 0, 40, 200 and 400 mg/kg/day (picloram acid equivalents) during days 6 to 18 of gestation. The maternal no observed adverse effect level (NOAEL) is 40 (34) mg/kg/day, where the lowest observed adverse effect level (LOAEL) is 200 (172) mg/kg/day based on reduced maternal weight gain during gestation. The developmental NOAEL is 400 (340) mg/kg/day and the LOAEL was not determined. The potassium salt of picloram was administered to CD rats by gastric intubation at dosage levels of 0, 35 (30), 174 (150) and 347 (298) mg/kg/day during day 6-15 of gestation: The test vehicle was distilled water. There was no evidence of developmental toxicity at doses up to and including the high dose of 347 (298) mg/kg/day. The maternal LOAEL is 347 (298) mg/kg/day based upon excessive salivation in the dams of the high dose group. Hence, the developmental toxicity NOAEL is greater than or equal to 347 (298) mg/kg/day. The maternal toxicity LOAEL is 347 (298) mg/kg/day and NOAEL is 174 (150) mg/kg/day.

Picloram acid was evaluated in a 2-generation reproduction study in the CD rat. Dosage levels employed were 0, 20, 200 or 1,000 mg/kg/day. The parental LOAEL is 1,000 mg/kg/day based on histopathological lesions in the kidney of males of both generations and some females. In males of both generations, blood in the urine, decreased urine specific gravity, increased absolute and relative kidney weight, and increased body weight gain was observed at the high dose. The parental LOAEL is 1,000 mg/kg/day and the NOAEL is 200 mg/kg/day. The reproductive LOAEL was not identified and the NOAEL is 1,000 mg/kg/day.

4. *Subchronic toxicity*. In a 90 day oral toxicity study, picloram acid was administered via the diet to groups of 15 F344 rats/sex/dose at dosage levels of 0, 15, 50, 150, 300 or 500 mg/kg/day. Based upon liver weight changes and minimal microscopic changes in the liver, the systemic LOAEL is 150 mg/kg/day. The NOAEL is 50 mg/kg/day.

In a 1982 6 month dog dietary study, picloram acid was evaluated at dosage levels of 0, 7, 35 or 175 mg/kg/day. The systemic NOAEL is 35 mg/kg/day and the LOAEL is 175 mg/kg/day based on decreases in body weight gain and food consumption and increases in liver weights (relative), alkaline phosphatase and alanine transaminase. Increased liver to body weight ratios and absolute liver weights were observed in only two males at the 35 mg/kg/day dosage level.

In a 21 day dermal toxicity study, the potassium salt of picloram was administered dermally to groups of five New Zealand white rabbits of each sex at doses of 0 (vehicle control), 75.3, 251 or 753 mg/kg/day (0, 65, 217 or 650 mg/kg/day picloram acid equivalents) for a total of 15 applications over the 21 day period. The NOAEL is greater than or equal to 753 mg/kg/day for both sexes: hence, a LOAEL was not established for either sex. Although the limit dose of 1,000 mg/kg/day was not achieved, practical difficulties precluded administering more test material. The study revealed the non-systemic effects of dermal irritation and very slight to well defined edema and/or erythema in both sexes at all dose levels.

5. *Chronic toxicity*. In a 1988 1 year chronic feeding study in the dog, picloram acid was administered orally via the diet at dosage levels of 0, 7, 35 or 175 mg/kg/day The LOAEL is 175 mg/kg/day based on increased liver weight (absolute and relative). The NOAEL is 35 mg/kg/day.

In a chronic toxicity/carcinogenicity feeding study conducted in the F344 rat, picloram acid (technical grade 93 % containing 197 ppm hexachlorobenzene as an impurity) was evaluated at 0, 20, 60 or 200 mg/kg/day for 2 years. The chronic toxicity LOAEL was 60 mg/kg/day as evidenced by altered size, tinctorial properties of centrilobular hepatocytes, and increased absolute and/or relative liver weights in both sexes. The NOAEL was 20 mg/kg/day. The study was negative for carcinogenicity, but due to concerns that a MTD may not have been achieved and the fact that the test material contained 197 ppm hexachlorobenzene impurity, the study was not considered to fulfill adequately the carcinogenicity testing requirement.

In response to the deficiencies cited in the study above, an additional 2 year dietary chronic/carcinogenicity study was conducted (in 1992) using F344 rats administered picloram acid at dosage levels of 0, 250 or 500 mg/kg/day for 104 weeks. Chronic toxicity was observed at 250 mg/kg/day among males only (increased incidence and severity of glomerulonephritis, blood in urine, decreased specific gravity of urine, increased size of hepatocytes that often had altered staining properties). Among females there were chronic effects only at 500 mg/kg/day (increased glomerulonephropathy, increased absolute and relative kidney weight). There was no evidence of carcinogenicity in this study. It should be noted that use of the Osborne-Mendel rat was waived due to lack of availability of the strain of rat. In addition, the level of hexachlorobenzene in the test material employed in this study was 12 ppm. These two studies fulfill the guidelines 83-l(a) and 83-2(a) for rats.

In a 1992 2 year dietary carcinogenicity study in B6C3F1 mice, picloram acid was evaluated at doses of 0, 100, 500 or 1,000 mg/kg/day. The systemic NOAEL in this study is 500

mg/kg/day based on a significant increase in absolute and relative kidney weights in males at the high dose level (HDT). No histopathological lesions were found to corroborate these changes. There was no evidence of carcinogenicity.

The dose levels tested in the 1992 carcinogenicity studies in rats and mice were considered adequate for carcinogenicity testing. The treatment did not alter the spontaneous tumor profile in mice or different strains of rats tested under the testing conditions. The chemical was classified as a ''Group E - Evidence of Non-Carcinogenicity for humans''. This classification applies to the picloram acid and potassium salt forms for which acceptable carcinogenicity studies were available for review by the HED Carcinogenicity Peer Review Committee (May 26, 1988).

Using its Guidelines for Carcinogen Risk Assessment published September 24, 1986 (51 FR 33992), picloram is classified as Group ''E'' for carcinogenicity (no evidence of carcinogenicity) based on the results of the carcinogenicity studies. The dose levels tested in the 1992 carcinogenicity studies in rats and mice were considered adequate for carcinogenicity testing. The treatment did not alter the spontaneous tumor profile in mice or different strains of rats tested under the testing conditions. The chemical was classified as a ''Group E - Evidence of Non-Carcinogenicity for humans''. This classification applies to the picloram acid and potassium salt forms for which acceptable carcinogenicity studies were available for review by the HED Carcinogenicity Peer Review Committee (May 26, 1988). Thus, a cancer risk assessment would not be appropriate.

Hexachlorobenzene (HCB), a recognized impurity in picloram compounds, is considered to be an animal carcinogen and probable human carcinogen as discussed in the 1988 Registration Standard for picloram. The Q* is 1.02 (mg/kg/day)-1. The maximum level of HCB in picloram is considered to be 0.005%.

6. *Animal metabolism*. The absorption, distribution, metabolism and excretion of picloram acid was evaluated in female rats administered a single i.v. or oral gavage dose of 10 mg/kg, an oral gavage dose of 1,000 mg/kg ^{14}C-picloram, or 1 mg/kg/day unlabeled picloram by gavage for 14 days followed by a single oral gavage dose of 10 mg/kg ^{14}C-picloram on day 15. The study demonstrates that ^{14}C-picloram is rapidly absorbed, distributed and excreted following oral and i.v. administration. This study alone is not adequate; however, this study is acceptable when considered in conjunction with a male rat metabolism study which yielded similar results.

7. *Endocrine disruption*. An evaluation of the potential effects on the endocrine systems of mammals has not been determined; However, no evidence of such effects were reported in the chronic or reproductive toxicology studies described above. There was no observed pathology of the endocrine organs in these studies. There is no evidence at this time that picloram causes endocrine effects.

C. Aggregate Exposure

In examining aggregate exposure, FFDCA section 408 requires that EPA take into account available and reliable information concerning exposure from the pesticide residue in the food in question, residues in other foods for which there are tolerances, residues in groundwater or surface water that is consumed as drinking water, and other non-occupational exposures through pesticide use in gardens, lawns, or buildings (residential and other indoor uses).

1. *Dietary exposure*—i. *Food*. For purposes of assessing the potential dietary exposure under these tolerances, aggregate exposure is estimated based on the theoretical maximum residue contribution (TMRC) from the existing and future potential tolerances for picloram on food crops. The TMRC is obtained by multiplying the tolerance level residues (existing and proposed) by the consumption data which estimates the amount of those food products eaten by various population subgroups. Exposure of humans to residues could also result if such residues are transferred to meat, milk, poultry or eggs. The following assumptions were used in conducting the HED exposure assessment 100% of the crops were treated, the RAC residues would be at the level of the tolerance, and some refinements were made based on marketing information previously supplied to HED by BEAD. This screening level analysis results in an overestimate of human exposure and a conservative assessment of risk. .

The chronic dietary exposure/risk estimates for picloram are extremely low. For the United States population as a whole, the TMRC is 0.0011 milligram kilogram body weight day (mg/kg/bwt/day), <1 of the RfD. The subgroup with the greatest routine chronic exposure is Non-nursing Infants (< 1 year old), which has a TMRC of 0.0042 mg/kg/bwt/day (2% of the RfD).

There is currently no form of sorghum observed in human consumption surveys utilized by EPA in their dietary risk evaluation system (DRES) assessments. Furthermore, residues of picloram in sorghum do not increase the dietary burden of picloram in animal feeds. Therefore, sorghum tolerances will have no effect on the human dietary consumption of picloram, and the proposed action, as well as existing tolerances, pose no concern with regards to chronic dietary exposure to food residues of picloram.

The estimated carcinogenic dietary risk for HCB as an impurity in picloram only for the U.S. population is 1.5 x10^{-7} which is less than the 1.0 x10^{-6} point below which risk is generally considered to be negligible.

ii. *Drinking water*. An additional potential source of dietary exposure to residues of pesticides are residues in drinking water. The Maximum Contaminant Level (MCL) for residues of picloram in drinking water has been established at 500 µg/L and a 1-10 day Health Advisory of 20,000 µg/L.

The Agency has published screening methods for estimating chemical residues in both ground water (SCI-GROW2) and surface water (GENEEC). Employing these methods yields the following 56 day Expected Environmental Concentrations (EEC) for a range of application rates:

Application rate (lb. acid equivalent/acre) and use	SCI-GROW2 EEC (µg/L)	GENEEC EEC (µg/L)
0.023 (wheat, barley, and oats use rate).	4.4	1.2
1 (maximum broadcast rate in label).	189	51.3
2 (maximum spot treatment rate in label).	379	103.1

The 56 day value is an appropriate endpoint to employ for the chronic exposure scenario. Default, conservative inputs were used for the models, as described in July 27, 1998 memorandum from EPA to Dow AgroSciences. Employing these values, a worst-case drinking water risk assessment can be performed as summarized below:

Population Subgroup[1]	RfD (mg/kg/day)	Food Exposure (mg/kg/day)	Maximum Water Exposure (mg/kg/day)[2]	DWLOC (μg/L)[3]	SCI-GROW2 EEC (μg/L)	GENEEC EEC (μg/L)
US Population	0.2	0.0011	0.2	7000	379	103.1
Females (13-19, not nursing or pregnant).	0.2	0.00090	0.2	6000	379	103.1
Non-Nursing infants (<< 1 yr. old).	0.2	0.0043	0.2	2000	379	103.1

[1] Population subgroups chosen in EPA memorandum of 7/27/98
[2] = RfD - ARC from DRES (cited above)
[3] Drinking water level of concern, based on default water body weights and water consumption of : 70 kg/2L (adult males), 60 kg/2L (adult female), 10 kg/1L (infant)

This tables shows that for even the most highly exposed population, exsure from water is below HED's DWLOC for chronic dietary exposure. Further refinement is also possible, based on monitoring data. Monitoring data available from the Pesticides in Ground Water Database indicate that picloram has been detected in ground water at concentrations ranging up to 30 μg/L. Results reported in this database typically were focused on highly vulnerable areas and in many cases, the database reports information from poorly constructed or damaged wells. These wells are at high risk because of the potential for surface residues to be carried directly down the casing into the ground water. Recognizing these high risk situations, an analysis of this database shows that less than 3% of the wells sampled were found to contain picloram. No distinction has been made between point and non point sources of material. Many of the detections are known to be related to point source contamination including spills at mixing/loading sites, near wells and back siphoning events. Of the detections which may have resulted from non-point sources, none are documented to occur on sites where application would be recommended based on current labeling. Nearly 99% of the ground water detections are at levels of less than 1% of the Maximum Contaminant Level (i.e., > 5 μg/L) established for human consumption by the EPA Office of Drinking Water. The STORET database maintained by the USEPA Office of Drinking Water indicates that picloram has been reported in surface water samples before 1988. Of these detections, 85% were at concentrations 0.13 μγ/L or lower and the maximum was 4.6 μγ/L. The maximum concentration reported was 4.6 μγ/L. Comparing these values to the DWLOC shows an even greater degree of protection for all of the population subgroups.

HCB contamination of ground water resources is relatively unlikely due to its high binding potential.

Based on monitoring data and fate properties it is unlikely that long term HCB concentrations in surface water would exceed 10 parts per trillion (ppt). Therefore, exposure from water is below EPA's drinking water level of concern of 34 ppt for chronic dietary exposure to HCB for the U.S. population.

In summary, these data on potential water exposure indicate insignificant additional dietary intake and risk for picloram.

2. *Non-dietary exposure*. This is a restricted use chemical that has no residential uses at this time; therefore, there are no human risks associated with residential uses. Entry into a treated area soon after the application of picloram is expected to be rare given the cultural practices typically associated with the use-sites (rights-of-way, forestry, pastures, range lands, and small grains) defined by the picloram labels at this time. Furthermore, if entry should occur, the potential exposures are expected to be minimal due to the characteristics of those use-sites

D. Cumulative Effects

Picloram is a pyridine carboxylic acid herbicide. Other herbicides in this class include clopyralid, quinclorac and thiazopyr. Section 408(b)(2)(D)(v) of the Food Quality Protection Act (FQPA) requires that, when considering whether to establish, modify, or revoke a tolerance, the Agency consider ''available information'' concerning the cumulative effects of a particular pesticide's residues and ''other substances that have a common mechanism of toxicity''. The Agency believes that ''available information'' in this context might include not only toxicity, chemistry, and exposure data, but also scientific policies and methodologies for understanding common mechanisms of toxicity and conducting cumulative risk assessments. For most pesticides, although the Agency has some information in its files that may turn out to be helpful in eventually determining whether a pesticide shares a common mechanism of toxicity with any other substances, EPA does not at this time have the methodologies to resolve the complex scientific issues concerning common mechanism of toxicity in a meaningful way. EPA has begun a pilot process to study this issue further through the examination of particular classes of pesticides. The Agency hopes that the results of this pilot process will increase the Agency's scientific understanding of this question such that EPA will be able to develop and apply scientific principles for better determining which chemicals have a common mechanism of toxicity and evaluating the cumulative effects of such chemicals. The Agency anticipates, however, that even as its understanding of the science of common mechanisms increases, decisions on specific classes of chemicals will be heavily dependent on chemical specific data, much of which may not be presently available.

Although at present the Agency does not know how to apply the information in its files concerning common mechanism issues to most risk assessments, there are pesticides as to which the common mechanism issues can be resolved. These pesticides include pesticides that are toxicologically dissimilar to existing chemical substances (in which case the Agency can conclude that it is unlikely that a pesticide shares a common mechanism of activity with other substances) and pesticides that produce a common toxic metabolite (in which case common mechanism of activity will be assumed).

EPA does not have, at this time, available data to determine whether picloram has a common mechanism of toxicity with other substances or how to include this pesticide in a cumulative risk assessment. For the purposes of these tolerance actions, therefore, EPA has not assumed that picloram has a

common mechanism of toxicity with other substances.

E. Safety Determination

1. *U.S. population*. In the meeting of September 30, 1993, the OPP RfD Peer Review Committee recommended that the RfD for this chemical be based on a NOAEL of 20 mg/kg/day for a dose-related increase in size and altered tinctorial properties of centrilobular hepatocytes in males and females at 60 and 200 mg/kg/day in a chronic toxicity study in rats. An uncertainty factor (UF) of 100 was used to account for the inter-species extrapolation and intra-species variability. On this basis, the RfD was calculated to be 0.20 mg/kg/day. The TMRC from existing tolerances is 0.001845 mg/kg/day. Existing tolerances utilize >1% of the RfD. It should be noted that no regulatory value has been established for this chemical by the World Health Organization (WHO) up to this date. The committee classified picloram as a ''Group E'' chemical, no evidence of carcinogenicity for humans.

Using the conservative exposure assumptions described above and based on the completeness and reliability of the toxicity data, it is concluded that aggregate exposure to picloram will utilize approximately 1% of the RfD for the U.S. population. Generally, exposures below 100% of the RfD are of no concern because the RfD represents the level at or below which daily aggregate dietary exposure over a lifetime will not pose appreciable risk to human health. Thus, there is a reasonable certainty that no harm will result from aggregate exposure to picloram residues.

2. *Infants and children*. In assessing the potential for additional sensitivity of infants and children to residues of picloram, data from developmental toxicity studies in the rat and rabbit and a 2-generation reproduction study in the rat were considered. The developmental toxicity studies are designed to evaluate adverse effects on the developing organism during prenatal development resulting from pesticide exposure to one or both parents. Reproduction studies provide (1) information relating to effects from exposure to the pesticide on the reproductive capability of mating animals and (2) data on systemic toxicity.

Developmental toxicity was studied using rats and rabbits. The developmental study in rats resulted in a developmental NOAEL of >298 mg/kg/day and a maternal toxicity NOAEL of 280 mg/kg/day. A study in rabbits resulted in a maternal NOAEL of 34 mg/kg/day and a developmental NOAEL of 344 mg/kg/day. Based on all of the data for picloram, there is no evidence of developmental toxicity at dose levels that do not result in maternal toxicity.

In a 2-generation reproduction study in rats, The NOAEL for parental systemic toxicity is 200 mg/kg/day. There was no effect on reproductive parameters at 1,000 mg/kg/day nor was there an adverse effect on the morphology, growth or viability of the offspring; thus, the reproductive NOAEL is 1,000 mg/kg/day.

FDCA section 408 provides that EPA may apply an additional safety factor for infants and children in the case of threshold effects to account for pre- and post-natal toxicity and the completeness of the database. Based on the current toxicological data requirements, the database relative to pre- and post-natal effects for children is complete. Therefore, it is concluded that an additional uncertainty factor is not warranted and that the RfD at 0.2 mg/kg/day is appropriate for assessing aggregate risk to infants and children.

Using the conservative exposure assumption previously described, it is concluded that the percent of the RfD that will be utilized by aggregate exposure to residues of picloram will be less than 4% of the RfD for all populations and subgroups. Since this estimate represents the 'worst case' exposure for a given population (Non-nursing infants, >1 year old), exposures will be less for all other sub-populations e.g. children, 1-6 years. Therefore, based on the completeness and reliability of the toxicity data and the conservative exposure assessment, it is concluded that there is a reasonable certainty that no harm will result to infants and children from aggregate exposure to picloram residues.

F. International Tolerances

There are no Codex maximum residue levels established for residues of picloram.

G. Other Considerations

Data Gaps. Residue data for sorghum aspirated grain fractions is currently being generated. Based on the toxicological data and the levels of exposure, EPA has determined that the proposed tolerances will be safe.

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ENVIRONMENTAL PROTECTION AGENCY

[PF–832; FRL–6027–6]

Notice of Filing of Pesticide Petitions

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice.

SUMMARY: This notice announces the initial filing of pesticide petitions proposing the establishment of regulations for residues of certain pesticide chemicals in or on various food commodities.

DATES: Comments, identified by the docket control number PF–832, must be received on or before December 21, 1998.

ADDRESSES: By mail submit written comments to: Public Information and Records Integrity Branch (7502C), Information Resources and Services Division, Office of Pesticides Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460. In person bring comments to: Rm. 119, CM #2, 1921 Jefferson Davis Highway, Arlington, VA.

Comments and data may also be submitted electronically to: opp-docket@epamail.epa.gov. Follow the instructions under ''SUPPLEMENTARY INFORMATION.'' No confidential business information should be submitted through e-mail.

Information submitted as a comment concerning this document may be claimed confidential by marking any part or all of that information as ''Confidential Business Information'' (CBI). CBI should not be submitted through e-mail. Information marked as CBI will not be disclosed except in accordance with procedures set forth in 40 CFR part 2. A copy of the comment that does not contain CBI must be submitted for inclusion in the public record. Information not marked confidential may be disclosed publicly by EPA without prior notice. All written comments will be available for public inspection in Rm. 119 at the address given above, from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays.

FOR FURTHER INFORMATION CONTACT: By mail: Bipin Gandhi, Registration Division (7505C), Office of Pesticide Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460. Office Location, telephone number, and e-mail address: Rm. 707A, CM #2 1921 Jefferson Davis Hwy., Arlington, VA 22202, (703–8380, e-mail: gandhi.bipin@epamail.epa.gov.

SUPPLEMENTARY INFORMATION: EPA has received pesticide petitions as follows