compared to those described in PP 7F4842.

G. Existing Tolerances

Existing tolerances have been established for L-glutamic acid, 40 CFR part 180.1187.

H. International Tolerances

No Codex maximum residue levels have been established for L-glutamic acid.

PP 7F4843

In the **Federal Register** of October 29, 1997 (62 FR 57170, FRL–5751–3) EPA issued a notice pursuant to section 408 of the FFDCA, 21 U.S.C. 346a(e) announcing the filing of a pesticide tolerance petition (PF–772) by Auxein Corporation. This notice included a summary of the petition prepared by the petitioner and this summary contained conclusions and arguments to support its conclusion that the petition complied with the FQPA of 1996. This petition requested that 40 CFR 180 be amended by establishing an exemption from the requirement of a tolerance for residues of the biological pest control agent gamma aminobutyric acid in or on all food commodities. The final rule exempted the biochemical gamma aminobutyric acid from the requirement of a tolerance on all food commodities when used as a plant growth enhancer in accordance with good agricultural practices. EPA published a final rule establishing a tolerance exemption in the **Federal Register** on January 7, 1998 (63 FR 676–679) (FRL–5764–5) amending 40 CFR 1180.1188. Recent research performed on this active ingredient indicates the method of protection is not restricted to growth enhancement, and Auxein Corporation wishes to delete the wording ''when used as a plant growth enhancer'' from the present exemption.

A. Product Name and Proposed Use Practices

AuxiGro WP Plant Metabolic Primer. When used as directed, AuxiGro has been shown to increase yields and/or quality of treated commodities, early ripening in certain vegetables, increased root growth, early flowering and fruit set, faster seed germination and rooting.

B. Product Identity/Chemistry

1. *Identity of the pesticide and corresponding residues.* Supporting data for this section were submitted with PP 7F4843. Supporting product chemistry data for the end-use product, AuxiGro WP (EPA Reg. No. 70810–1) were submitted on June 12, 1997 (MRID 44296801) and February 16, 1998 (MRID 44538701).

2. *Magnitude of residue at the time of harvest and method used to determine the residue.* Supporting data for this section were submitted with PP 7F4843.

3. *A statement of why an analytical method for detecting and measuring the levels of the pesticide residue are not needed.* Supporting data for this section were submitted with PP 7F4843.

C. Mammalian Toxicological Profile

Supporting data for this section were submitted with PP 7F4843.

D. Aggregate Exposure

1. *Dietary exposure*—i. *Food.* No differences in exposure are expected compared to those described in PP 7F4843.

ii. *Drinking water.* No differences in exposure are expected compared to those described in PP 7F4843.

2. *Non-dietary exposure.* No differences in exposure are expected compared to those described in PP 7F4843.

E. Cumulative Exposure

No differences in exposure are expected compared to those described in PP 7F4843.

F. Safety Determination

1. *U.S. population.* Based on its abundance in nature and long history of use by humans without deleterious effects, there is reasonable certainty that no harm will result from aggregate exposure to the U.S. population, including infants and children, to residues of GABA. This includes all anticipated dietary exposure and all other exposures for which there is reliable information. This is a reasonable conclusion because exposure to GABA resulting from label directed use is inconsequential, does not cross the blood-brain barrier, and is consumed daily by the human population from naturally occurring sources.

2. *Infants and children.* No differences in exposure are expected compared to those described in PP 7F4843.

G. Existing Tolerances

Existing tolerances have been extablished for gamma aminobutyric acid (GABA), 40 CFR part 180.1188.

H. International Tolerances

No Codex maximum residue levels have been established for gamma aminobutyric acid (GABA)

[FR Doc. 00–30918 Filed 12–5–00; 8:45a.m.]

BILLING CODE 6560–50–S

ENVIRONMENTAL PROTECTION AGENCY

[PF–985; FRL–6755–5]

Notice of Filing a Pesticide Petition to Establish a Tolerance for a Certain Pesticide Chemical in or on Food

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 docket control number PF–985, must be received on or before January 5, 2001.

ADDRESSES: Comments may be submitted by mail, electronically, or in person. Please follow the detailed instructions for each method as provided in Unit I.C. of the **SUPPLEMENTARY INFORMATION.** To ensure proper receipt by EPA, it is imperative that you identify docket control number PF–985 in the subject line on the first page of your response.

FOR FURTHER INFORMATION CONTACT: By mail: James Tompkins, Registration Support Branch, Registration Division (7505C), Office of Pesticide Programs, Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460; telephone number: (703) 305–5697; e-mail address: tompkins.jim@epa.gov.

SUPPLEMENTARY INFORMATION:

I. General Information

A. Does this Action Apply to Me?

You may be affected by this action if you are an agricultural producer, food manufacturer or pesticide manufacturer. Potentially affected categories and entities may include, but are not limited to:

Categories	NAICS codes	Examples of potentially affected entities
Industry	111 112 311 32532	Crop production Animal production Food manufacturing Pesticide manufacturing

This listing is not intended to be exhaustive, but rather provides a guide for readers regarding entities likely to be affected by this action. Other types of entities not listed in the table could also be affected. The North American Industrial Classification System

(NAICS) codes have been provided to assist you and others in determining whether or not this action might apply to certain entities. If you have questions regarding the applicability of this action to a particular entity, consult the person listed under **FOR FURTHER INFORMATION CONTACT.**

B. How Can I Get Additional Information, Including Copies of this Document and Other Related Documents?

1. *Electronically*. You may obtain electronic copies of this document, and certain other related documents that might be available electronically, from the EPA Internet Home Page at http://www.epa.gov/. To access this document, on the Home Page select ''Laws and Regulations'', and then look up the entry for this document under the ''**Federal Register**—Environmental Documents.'' You can also go directly to the **Federal Register** listings at http://www.epa.gov/fedrgstr/.

2. *In person*. The Agency has established an official record for this action under docket control number PF–985. The official record consists of the documents specifically referenced in this action, any public comments received during an applicable comment period, and other information related to this action, including any information claimed as confidential business information (CBI). This official record includes the documents that are physically located in the docket, as well as the documents that are referenced in those documents. The public version of the official record does not include any information claimed as CBI. The public version of the official record, which includes printed, paper versions of any electronic comments submitted during an applicable comment period, is available for inspection in the Public Information and Records Integrity Branch (PIRIB), Rm. 119, Crystal Mall #2, 1921 Jefferson Davis Highway, Arlington, VA, from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays. The PIRIB telephone number is (703) 305–5805.

C. How and to Whom Do I Submit Comments?

You may submit comments through the mail, in person, or electronically. To ensure proper receipt by EPA, it is imperative that you identify docket control number PF–985 in the subject line on the first page of your response.

1. *By mail*. Submit your comments to: Public Information and Records Integrity Branch (PIRIB), Information Resources and Services Division (7502C), Office of Pesticide Programs (OPP), Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460.

2. *In person or by courier*. Deliver your comments to: Public Information and Records Integrity Branch (PIRIB), Information Resources and Services Division (7502C), Office of Pesticide Programs (OPP), Environmental Protection Agency, Rm. 119, Crystal Mall #2, 1921 Jefferson Davis Highway, Arlington, VA. The PIRIB is open from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays. The PIRIB telephone number is (703) 305–5805.

3. *Electronically*. You may submit your comments electronically by e-mail to: opp-docket@epa.gov, or you can submit a computer disk as described above. Do not submit any information electronically that you consider to be CBI. Avoid the use of special characters and any form of encryption. Electronic submissions will be accepted in Wordperfect 6.1/8.0 or ASCII file format. All comments in electronic form must be identified by docket control number PF–985. Electronic comments may also be filed online at many Federal Depository Libraries.

D. How Should I Handle CBI That I Want to Submit to the Agency?

Do not submit any information electronically that you consider to be CBI. You may claim information that you submit to EPA in response to this document as CBI by marking any part or all of that information as CBI. Information so marked will not be disclosed except in accordance with procedures set forth in 40 CFR part 2. In addition to one complete version of the comment that includes any information claimed as CBI, a copy of the comment that does not contain the information claimed as CBI must be submitted for inclusion in the public version of the official record. Information not marked confidential will be included in the public version of the official record without prior notice. If you have any questions about CBI or the procedures for claiming CBI, please consult the person identified under **FOR FURTHER INFORMATION CONTACT.**

E. What Should I Consider as I Prepare My Comments for EPA?

You may find the following suggestions helpful for preparing your comments:

1. Explain your views as clearly as possible.

2. Describe any assumptions that you used.

3. Provide copies of any technical information and/or data you used that support your views.

4. If you estimate potential burden or costs, explain how you arrived at the estimate that you provide.

5. Provide specific examples to illustrate your concerns.

6. Make sure to submit your comments by the deadline in this notice.

7. To ensure proper receipt by EPA, be sure to identify the docket control number assigned to this action in the subject line on the first page of your response. You may also provide the name, date, and **Federal Register** citation.

II. What Action is the Agency Taking?

EPA has received a pesticide petition as follows proposing the establishment and/or amendment of regulations for residues of a 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 support granting of the petition. Additional data may be needed before EPA rules on the petition.

List of Subjects

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

Dated: November 21, 2000.

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 view of the petitioners. EPA is publishing the petition summary verbatim without editing it 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.

Dow AgroSciences

PP 4F4412

EPA has received a 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 (FFDCA), 21 U.S.C. 346a(d), to amend 40 CFR part 180 by extending the time–limited tolerance for residues of picloram, 4-amino-3,5,6-trichloropicolinic acid and its potassium salt in or on in or on the raw agricultural commodities (RACs) sorghum grain at 0.3 parts per million (ppm), sorghum grain forage at 0.2 ppm, and sorghum stover at 0.5 ppm; and for residues of picloram in or on the RAC aspirated grain fractions at 4 ppm until December 31, 2002. EPA issued a final rule, published in the **Federal Register** of January 5, 1999 (64 FR 418) (FRL–6039–4), which announced that it established a time–limited tolerance for the indirect or inadvertent residues of the herbicide picloram, 4-amino-3,5,6-trichloropicolinic acid and its potassium salt in or on sorghum grain at 0.3 ppm, sorghum grain forage at 0.2 ppm, and sorghum stover at 0.5 ppm; and for residues of picloram in or on the RAC aspirated grain fractions at 4 ppm, with an expiration date of December 31, 2000. A condition of this rule required Dow AgroSciences to submit an aspirated grain residue study before December 31, 1999, which they did on December 9, 1999. The extension of the time–limited tolerances to December 31, 2002 will allow time for review of this additional data and establishment of final tolerances. EPA has determined that the petition contains data or information regarding the elements set forth in section 408(d)(2) of the FFDCA; 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*

TABLE 1.–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
Aspirated grain fractions	ND

[a] ND = less than one–half of the validated lower limit of quantitation of 0.05 µg/g in grain, 0.1 µg/g in forage and fodder, and 0.25 µg/g in aspirated grain fractions.

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 milligram/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 milligram/liter (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 µ g/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 gram/milliliter (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 g/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 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 (vehicle control) 0, 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 two years. The chronic toxicity LOAEL was 60 mg/kg/day as evidenced by altered size and 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). 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

1. *Dietary exposure.*–i. *Food.* For purposes of assessing the potential

dietary exposure under these tolerances, aggregate exposure is estimated based on the 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 Theoretical Maximum Residue Contribution (TMRC) is 0.0011 mg/kg bw/day, < 1 of the reference dose (RfD). The subgroup with the greatest routine chronic exposure is Non–nursing Infants (Less Than 1–Year Old), which has a TMRC of 0.0042 mg/kg bw/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 model (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 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:

TABLE 2.–EXPECTED ENVIRONMENTAL CONCENTRATIONS

Application rate (lb. acidequivalent/acre) and use	SCI–GROW2EEC (µg/L)	GENEECEEC (µg/L)
0.023 (wheat, barley,and oats use rate)	4.4	1.2
1 (maximum broadcastrate in label)	189	51.3
2(maximum spottreatment 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:

TABLE 3.–DRINKING WATER RISK ASSESSMENT

Population subgroup[1]	RfD (mg/kg/day)	Food exposure (mg/kg/day)	Maximum water exposure(mg/kg/day)[2]	DWLOC(µg/L)[3]	SCI–GROW^{2EEC} (µg/L)	GENEEC EEC (µg/L)
U.S population	0.2	0.0011	0.2	7,000	379	103.1
Females(13–19, not nursing orpregnant)	0.2	0.00090	0.2	6,000	379	103.1
Non–Nursing infants(< 1 yr. old)	0.2	0.0043	0.2	2,000	379	103.1

[1] Population subgroups chosen in EPA memorandum of July/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, exposure 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 Data base 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 data base 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 μg/L or lower and the maximum was 4.6 μg/L. The maximum concentration reported was 4.6 μg/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 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

The potential for cumulative effects of picloram and other substances that have a common mechanism of toxicity was considered. The mammalian toxicity of picloram is well defined. However, the biochemical mechanism of toxicity of this compound is not well known. No reliable information exists to indicate that toxic effects produced by picloram would be cumulative with those of any other chemical compounds. Therefore, consideration of a common mechanism of toxicity with other compounds is not appropriate. Thus, only the potential risks of picloram are considered in the aggregate exposure assessment.

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 theoretical maximum residue contribution (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 (i) information relating to effects from exposure to the pesticide on the reproductive capability of mating animals and (ii) 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.

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–natal and post–natal toxicity and the completeness of the data base. Based on the current toxicological data requirements, the data base relative to pre–natal and post–natal effects for children is complete. Therefore, it is concluded that an additional UF 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.

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ENVIRONMENTAL PROTECTION AGENCY

[PF–984; FRL–6755–4]

Notice of Filing a Pesticide Petition to Establish a Tolerance for a Certain Pesticide Chemical in or on Food

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 docket control number PF–984, must be received on or before January 5, 2001.

ADDRESSES: Comments may be submitted by mail, electronically, or in person. Please follow the detailed