1. What innovative approaches are being used (or could be used) by other environmental agencies, other regulatory agencies, and law enforcement agencies to measure the effects of their enforcement and compliance assurance programs?

2. What innovative approaches are being used by regulated facilities, companies, or trade groups and associations to measure the effect of their efforts to achieve and maintain compliance and protect the environment?

3. What can EPA use to measure the impact of its enforcement and compliance assurance program in low-income/minority population communities?

4. How can EPA measure industry performance in complying with environmental laws and regulations?

5. How can EPA measure the deterrent effect of its enforcement-related activities, including conducting inspections, taking enforcement actions, and publicizing those actions?

6. How can EPA measure the impact of compliance assistance activities and compliance incentives, such as its audit and self-disclosure policy?

EPA will use the upcoming stakeholders/regulatory partners meetings to further explore these issues.

III. Next Phase of the Strategy

As part of the Strategy, EPA now intends to meet with sets of stakeholders through the month of July to further discuss ideas and proposals for improved measures. Stakeholder participants will be asked to discuss guiding principles or specific measures that have been suggested to EPA at a prior public meeting or through independent submission. EPA will identify these discussion areas and circulate agenda items to participants or potential participants in advance of each meeting. Participants might be asked to prepare written comment on the specific issues and ideas identified in the meeting agenda and related materials.

These meetings will be open to the public, will be a half or full day in length, and will be limited to a maximum of 25 stakeholder participants. The meetings will take place in a ''roundtable'' format to promote interaction and more detailed discussion.

IV. Schedule of Stakeholders/ Regulatory Partners Meetings

Listed below is the schedule of meetings as currently developed by EPA. The schedule is subject to revision if necessary to avoid unforeseen conflicts or to accommodate additional meetings with stakeholders and regulatory partners.

(1) Wednesday, May 28, 1997, Federal Oversight Groups, (GAO, IG, OMB, and Congressional Appropriations Staff), 9:00 am—1:00 pm, Ariel Rios Building (Room #6045), 1200 Pennsylvania Avenue, NW, Washington, D.C.

(2) Thursday, May 29, 1997, Mixed Stakeholders, (Industry, Environmental and Environmental Justice Organizations), 9:00 am—5:00 pm, Washington, D.C., (Location to be determined)

(3) Wednesday, June 4, 1997, State Environmental Agencies 9:00 am—5:00 pm, EPA Region V–Chicago, IL, Great Lakes Conference Center (Lake Erie Room), 77 West Jackson Boulevard, Chicago, IL 60604–3507

(4) Thursday, June 12, 1997, Federal Regulatory Agencies, (FDA, OSHA, IRS, Customs, Coast Guard, etc.), 9:00 am—5:00 pm, Washington, D.C.

(5) Wednesday, June 25, 1997, Mixed Stakeholders, (Additional State Environmental Agencies, State AGs, Tribes, Media-Specific Associations, and Local Government Associations), 9:00 am—5:00 pm, (Location to be determined)

(6) Beginning of July (if necessary), Mixed Stakeholders, (Industry, Environmental and Environmental Justice Organizations), Washington, D.C.

(7) Late July or Beginning of August 1997, Meeting with House Staff, Meeting with Senate Staff, Second Meeting with Federal Oversight Groups

(8) Week of September 15, 1997, Capstone Conference in Washington, D.C.

V. Information for Participants

Parties interested in participating in these meetings should contact James McDonald at (202) 564–4043. In addition, EPA will be soliciting participants through various organizations and associations. Participants interested in more detailed information about the Strategy or the two public meetings, including transcripts and statements of stakeholders, can review documents at EPA's Information Resource Center, which is located at 401 M Street, SW (Room #M2904), Washington, DC 20460 (202) 260-5921, or access these documents on-line at EPA's EnviroSense web site. (The address is: http://es.inel.gov/oeca/perfmeas)

Dated: May 5, 1997.

Michael M. Stahl,

Deputy Assistant Administrator, Office of Enforcement and Compliance Assurance.

[FR Doc. 97–12477 Filed 5–12–97; 8:45 am]

BILLING CODE 6560–50–P

ENVIRONMENTAL PROTECTION AGENCY

[PF–731; FRL–5714–3]

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–731, must be received on or before June 12, 1997.

ADDRESSES: By mail submit written comments to: Public Information and Records Integrity Branch, Information Resources and Services Division (7506C), Office of Pesticides Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460. In person bring comments to: Rm. 1132, 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. 1132 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: Philip Errico, Product Manager (PM-25), Registration Division (7505C), Office of Pesticide Programs, Environmental Protection Agency, 401 M St., S.W., Washington, D.C. 20460.

Office Location, telephone number, and e-mail address: Rm. 241 Crystal Mall #2, 1921 Jefferson Davis Highway, Arlington, VA 22202, (703) 305–6800; e-mail: errico.phil@epamail.epa.gov.

SUPPLEMENTARY INFORMATION: EPA has received pesticide petitions as follows proposing the establishment and/or amendment of regulations for residues of certain pesticide chemicals 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 these petitions contain 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 grantinig 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–731] (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 file format or ASCII file format. All comments and data in electronic form must be identified by the docket number (insert docket number) and appropriate petition number. Electronic comments on this proposed rule 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: April 30, 1997.

James Jones,

Acting Director, Registration Division, Office of Pesticide Programs.

Summaries of Petitions

Petitioner summaries of the pesticide petitions are printed below as required by section 408(d)(3) of the FFDCA. The summaries of the petitions were prepared by the petitioners and represent the views of the petitioners. 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. DowElanco

PP 4F4412

EPA has received a pesticide petition (PP 4F4412) from DowElanco 9330 Zionsville Road Indianapolis, IN 46254 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 ppm, respectively. The proposed analytical method is ACR 73.3.S2. Pursuant to the sect 408(d)(2)(A)(i) of the FFDCA, as amended, Company has submitted the following summary of information, data and arguments in support of their pesticide petition. This summary was prepared by DowElanco and EPA has not fully evaluated the merits of the petition. EPA edited the summary to clarify that the conclusions and arguments were the petitioner's and not necessarily EPA's and to remove certain extraneous material.

Picloram provides control of deep rooted perennial weeds either in grainland, fallowland or on CRP acres. With the addition of the proposed tolerance, grain sorghum could be considered as a rotational crop option for the producer. The Agency has completed the reregistration review of picloram, culminating in publication of the Reregistration Eligibility Decision (RED) for picloram which was received on October 5, 1995. The RED concludes that picloram and its derivatives can be used without causing unreasonable adverse effects to humans or the environment. Therefore, all uses of products containing picloram acid and its derivatives were judged eligible for reregistration. In view of this comprehensive regulatory review, as well as the lack of human dietary consumption of grain sorghum and the negligible dietary impact on livestock associated with this proposed use, establishment of these tolerances will not cause exposure to exceed the levels at which there is an appreciable risk.

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 DowElanco 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 —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 per 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. *Genotoxicity*. Picloram acid was evaluated in the Ames test using Salmonella typhimurium. Doses ranged up to 5,000 ug/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 (750 ug/ml without metabolic activation; 1,250 ug/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 intragastric 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 ug/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 ug/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 Savage at dosage levels of 0, 40, 200 and 400 milligram per kilogram per day (mg/kg/day) (picloram acid equivalents) during days 6 to 18 of gestation. The maternal NOEL is 40 (34) mg/kg/day, where the LOEL is 200 (172) mg/kg/day based on reduced maternal weight gain during gestation. The developmental NOEL is 400 mg/kg/day and the LOEL 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 LOEL is 347 (298) mg/kg/day based upon excessive salivation in the dams of the high dose group. Hence, the developmental toxicity NOEL is greater than or equal to 347 (298) mg/kg/day. The maternal toxicity LOEL is 347 (298) mg/kg/day and NOEL 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 LOEL 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 LOEL is 1,000 mg/kg/day and the NOEL is 200 mg/kg/day. The reproductive LOEL was not identified and the NOEL 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 LOEL is 150 mg/kg/day. The NOEL 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 NOEL is 35 mg/kg/day and the LOEL is 175 mg/kg/day based on decreases in the following: body weight gain, food consumption, liver weights (relative), alkaline phosphatase and alanine transaminase. Increased liver to body weight ratios and absolute 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 (O. 65, 217 or 650 mg/kg/day picloram acid equivalents) for a total of 15 applications over the 21–day period. The NOEL is greater than or equal to 753 mg/kg/day for both sexes: hence, a LOEL 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 LOEL is 175 mg/kg/day based on increased liver weight (absolute and relative). The NOEL 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 LOEL 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 NOEL 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 NOEL 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 (5/26/88).

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 (5/26/88). Thus, a cancer risk assessment would not be appropriate.

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.

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 this exposure assessment: 100% of the crops were treated, the RAC residues would be at the level of the tolerance, and certain processed food residues would be at anticipated (average) levels based on processing studies (see attached Dietary Risk Evaluation for Picloram). This results in an overestimate of human exposure and a conservative assessment of risk. As mentioned previously, 0.9% of the RfD is utilized using these assumptions.

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 0.001845 milligrams per kilogram of body weight per day (mg/kg bw/day), only 0.9% of the RfD. For this same group, the Anticipated Residue Contribution (ARC) is 0.001053 mg/kg bw/day, only 0.5% of the RfD. The subgroup with the greatest routine chronic exposure/risk is non-nursing infants (less than 1 year old), which has a TMRC of 0.004753 mg/kg bw/day (2.4% of the RfD) and an ARC of 0.003805 mg/kg bw/day (1.9% of the RfD).

There is currently no form of sorghum observed in human consumption surveys utilized by EPA in their DRES assessments. 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.

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. 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 detection's are known to be related to point source contamination including spills at mixing/loading sites, near wells and back siphoning events. Of the detection's 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 detection's 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 µg/L or lower and the maximum was 4.6 µg/L. The maximum concentration reported was 4.6 µg/L.

The impact of potential residues of picloram in drinking water on the aggregate risk of the herbicide is minimal. If it is assumed that all of the drinking water in the U.S. contains 30 µg/L of picloram, the maximum observed in the groundwater data base, its contribution to the TMRC would be 0.000280 mg/kg bw/day for the general U.S. population, or 0.14% of the RfD. For the most sensitive population subgroup, Non-nursing Infants (<1 yr. old), the contribution to the TMRC would be 0.002855 mg/kg bw/day, or 1.4% of the RfD. In reality, the likelihood of drinking water being contaminated with picloram is extremely remote, and actual contribution to the dietary exposure of picloram is virtually nil.

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 NOEL 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 0.9% 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 percent of the RfD for the U.S. population. Generally, exposures below 100 percent 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 NOEL of >298 mg/kg/day and a maternal toxicity NOEL of 280 mg/kg/day. A study in rabbits resulted in a maternal NOEL of 34 mg/kg/day and a developmental NOEL 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 NOEL 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 NOEL 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- 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 percent 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.

Other Considerations

F. International Tolerances

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

1. *Endocrine effects*. 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.

2. *Data gaps*. Data gaps currently exist for residue data for sorghum aspirated grain fractions. Based on the toxicological data and the levels of exposure, EPA has determined that the proposed tolerances will be safe.

2. Novartis Crop Protection

PP 6F4688

EPA has received a pesticide petition (PP 6F4688) from Novartis Crop Protection, Inc., P. O. Box 18300, Greensboro, North Carolina 27419, 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 residues of the herbicide CGA-277476, Benzoic acid, 2-[[[[4,6-dimethyl-2-pyrimidinyl)-amino]carbonyl]amino]sulfonyl]-,3-oxetanylester in or on the raw agricultural commodity soybeans at 0.01 ppm. The proposed analytical method involves homogenization, filtration, partition and cleanup with analysis by high performance liquid chromatography using UV detection. 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.

As required by section 408(d) of the FFDCA, as recently amended by the Food Quality Protection Act (FQPA) Pub.L. 104-170, Novartis Crop Protection included in the petition a summary of the petition and authorization for the summary to be published in the **Federal Register** in a notice of receipt of the petition. The summary represents the views of Novartis Crop Protection. EPA is in the process of evaluating the petition. As

required by section 408(d)(3) of the FFDCA, EPA is including the summary as a part of this notice of filing. EPA has made minor edits to the summary for the purpose of clarity.

A. Metabolism

The qualitative nature of the metabolism of CGA-277476 in plants and animals is well understood for the purposes of the proposed tolerance. Metabolism proceeds through hydrolysis of the oxetane ring with subsequent cleavage of the oxetane ester and the sulfonylurea bridge. Metabolic pathways in plants (soybeans), rats, ruminants (goats), and poultry are similar. Parent CGA-277476 is the residue of concern.

B. Analytical Methodology

Novartis Crop Protection, Inc. has submitted a practical analytical method involving homogenization, filtration, partition and cleanup with analysis by high performance liquid chromatography using UV detection. The methodology accounts for residues of CGA-277476. The limit of quantitation (LOQ) for the method is 0.01 ppm for CGA-277476. This method has undergone a successful method trial and is available for enforcement.

C. Residue

Twenty field trials were conducted in typical soybean growing areas across the U.S. Either a single preplant or preemergence application (57 grams ai/A) or a split application made preemergence followed by a post broadcast application (total of 81 grams ai/A) was made. No residues (<0.01 ppm) were found in the dry beans (1X) and no residues were found in the processed commodities at rates up to 5X. No residues (<0.01 ppm) were found in rotational crops treated at the 1X rate. A prohibition against grazing forage, hay and silage will be placed in the label, as will a 60 day preharvest interval.

D. International MRL's

There are no Codex Alimentarius Commission (CODEX) maximum residue levels (MRL's) established for residues of CGA-277476 in or on raw agricultural commodities.

E. Toxicological Profile

1. *Acute toxicity*. CGA-277476 has a low order of acute toxicity. The rat oral LD_{50} is > 5,000 mg/kg, the acute rabbit dermal LD_{50} is > 2,000 mg/kg and the rat inhalation LC_{50} is > 5.08 mg/L. CGA 277476 is moderately irritating to the skin but not irritating to the eye. It is not a skin sensitizer in guinea pigs. The commercial formulation (75WG) of CGA-277476 has a similar acute toxicity profile, with both technical and formulated product carrying a Category III CAUTION Signal Word.

2. *Genotoxicity*. Assays for genotoxicity were comprised of tests evaluating the potential of CGA-277476 to induce point mutations (Salmonella assay and a Chinese hamster V79 lung tissue assay), chromosome aberrations (mouse micronucleus and a Chinese hamster ovary study) and the ability to induce either scheduled or unscheduled DNA synthesis in rat hepatocytes. The results indicate that CGA-277476 is not mutagenic or clastogenic and does not induce unscheduled DNA synthesis.

3. *Developmental/reproductive effects*. The developmental and teratogenic potential of CGA-277476 was investigated in rats and rabbits. The results indicate that CGA-277476 was not maternally or developmentally toxic in the rabbit. Minimal developmental toxicity was observed at the limit dose (1,000 mg/kg) in the rat; the developmental no observed effect level in the rat was 300 mg/kg/day. No evidence of teratogenicity was observed at the limit dose of 1,000 mg/kg in either the rat or rabbit.

A 2–generation reproduction study was conducted with CGA-277476 at feeding levels of 0, 20, 200, 5,000 or 20,000 ppm (0, 1, 10, 250 or 1,000 mg/kg/day). The reproductive NOEL was established at a feeding level of 5,000 ppm (equivalent to approximately 250 mg/kg/day). Reduced fertility observed at the highest dose tested (20,000 ppm) was associated with degenerative changes in the seminiferous tubules and atypical spematogenesis in males and severe effects on kidneys in females. The NOEL for parental toxicity was established at the 200 ppm feeding level based on slight effects on body weight parameters at the next highest dose tested (i.e. 5,000 ppm).

4. *Subchronic toxicity*. The subchronic toxicity of CGA-277476 was evaluated in studies in the rat, mouse and dog at high doses. Target organs included the liver, spleen, blood, kidney, urogenital tract, testes, epididymis and peripheral nerves and muscles. No observable effect levels have been established for all end-points in subchronic studies. The dog appears to be the most sensitive species (NOEL = 40 ppm; 1 mg/kg) with treatment related effects on testes, peripheral nerve and muscle appearing at doses ≥ 5,000 ppm (125 mg/kg/day).

5. *Chronic effects*. The chronic toxicity of CGA-277476 was investigated in long term studies in the rat, mouse and dog. Target organs included the central and peripheral nervous systems, skeletal muscle, liver, kidney, gallbladder, testes, and blood. No observed effect levels (NOELS) have been established in each study. The dog is the most sensitive species with a NOEL = 40 ppm (1.3 mg/kg/day). Based on these data, it is expected the EPA will establish a RfD for CGA-277476 at 0.01 mg/kg/day using the NOEL of 1.3 mg/kg/day and an uncertainty factor of 100.

6. *Carcinogenicity*. The carcinogenicity studies conducted with CGA- 277476 showed no evidence of an oncogenic response in either mouse or rat at doses that did not exceed the maximum tolerated dose. Dose levels in the mouse study were 2.25, 150, 525, and 1,050 mg/kg/day. In the rat study, dose levels were 1, 10, 100, 500, 750 (females), and 1,000 (males) mg/kg/day. At the end of the chronic rat study, a statistically significant increased incidence of schwannomas was found in the heart of the 1,000 mg/kg/day male rats (7/59) compared to the control group (0/60). Based on the Guidelines for Carcinogenic Risk Assessment published by EPA September 24, 1986 (51 FR 33992), Novartis Crop Protection believes that CGA-277476 should be classified as Class E because the neoplastic response (marginal increased incidence of schwannomas) was observed only in male rats at a dose exceeding the maximum tolerated dose of 500 mg/kg/day. No effect was observed at doses ≤ 500 mg/kg/day.

F. Threshold Effects

1. *Chronic effects*. Based on the available chronic toxicity data, it is expected the EPA will establish a RfD for CGA-277476 at 0.01 mg/kg/day based on the results obtained in the 1–year feeding study in dogs using the No-Observed Effect Level (NOEL) of 1.3 mg/kg/day and an uncertainty factor of 100.

2. *Acute toxicity*. Based on the available acute toxicity data, Novartis Crop Protection believes CGA-277476 does not pose any acute dietary risks.

G. Nonthreshold Effects.

Carcinogenicity. Based on the Guidelines for Carcinogenic Risk Assessment published by EPA September 24, 1986 (51 FR 33992), Novartis Crop Protection believes that CGA-277476 should be classified as Class E because the neoplastic response (marginal increased incidence of schwannomas) was observed only in male rats at a dose exceeding the maximum tolerated dose of 500 mg/kg/day. No effect was observed at doses ≤ 500 mg/kg/day.

H. Endocrine Effects.

CGA-277476 belongs to the sulfonylurea class of chemicals, one not known or suspected of having adverse effects on the endocrine system. Reduced fertility observed in high dose females (20,000 ppm) in the rat reproduction study was associated with degenerative changes in the seminiferous tubules and a typical spermatogenesis observed in high dose males. Evidence of impaired spermatogenesis was also observed at high doses (≥ 125 mg/kg/day) in the subchronic dog study.

I. Aggregate Exposure

1. *Dietary exposure*. For purposes of assessing the potential dietary exposure to CGA-277476, Novartis Crop Protection has estimated aggregate exposure based on the Theoretical Maximum Residue Contribution from the use of CGA-277476 in or on raw agricultural commodities for which tolerances have been proposed (0.01 ppm on soybeans). In conducting this exposure assessment, Novartis has conservatively assumed that 100% of soybeans will contain CGA-277476 residues at the proposed level of 0.01 ppm. No residues are anticipated in animal commodities and therefore, tolerances in meat, meat byproducts, milk, poultry and eggs are not proposed.

2. *Drinking water exposure*. Another potential source of exposure of the general population to residues of pesticides are residues in drinking water. The potential for CGA-277476 to enter surface or ground water sources of drinking water is limited because of the low use rate. This is supported by the results of two small-scale prospective ground water monitoring studies which did not show any quantifiable residues of CGA-277476 in ground water samples. The Maximum Contaminant Level Guideline (MCLG) calculated for CGA-277476 according to EPA's procedure leads to an exposure value (7 ppb) substantially greater than any level expected to reach ground water based on study results.

3. *Non-occupational exposure*. Novartis Crop Protection has evaluated the estimated non-occupational exposure to CGA-277476 and concludes that the potential for non-occupational exposure to the general population is unlikely because CGA-277476 is not planned to be used in or around the home, including home lawns, schools, recreation facilities or parks.

J. Cumulative Risk.

Novartis Crop Protection has also considered the potential for cumulative effects of CGA-277476 and other chemicals belonging to this chemical class (sulfonylureas) that may have a common mechanism of toxicity. It is concluded that consideration of a common mechanism of toxicity is not appropriate at this time because there is no reliable data to establish whether a common mechanism exists.

K. Safety Determinations.

1. *U.S. general population*. Using the conservative exposure assumptions described above, based on the completeness and reliability of the toxicity data, Novartis Crop Protection has concluded that aggregate exposure to CGA-277476 will utilize 0.07 percent of the RfD for the U.S. population based on chronic toxicity endpoints. Because EPA generally has no concern for exposures below 100 percent of the RfD, it is concluded that there is a reasonable certainty that no harm to the general population will result from aggregate exposure to CGA-277476.

2. *Infants and children*. In assessing the potential for additional sensitivity of infants and children to residues of CGA-277476, Novartis Crop Protection has considered data discussed above from developmental toxicity studies conducted with CGA-277476 in the rat and rabbit and a 2–generation rat reproduction study. The developmental toxicity studies are designed to evaluate adverse effects on the developing organism resulting from chemical exposure during prenatal development to one or both parents. Reproduction studies provide information relating to effects from exposure to a chemical on the reproductive capability of mating animals and data on systemic toxicity.

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 database. Based on the current toxicological data requirements, the database relative to pre- and post-natal effects for children is complete. Further, for CGA-277476, the NOEL of 1.3 mg/kg/day from the chronic dog study, which was used to calculate the RfD (discussed above), is at least an order of magnitude lower than the developmental NOEL of 300 mg/kg/day from the rat teratogenicity study or the reproductive NOEL of 250 mg/kg/day from the multigeneration reproduction study. There is no evidence to suggest that developing organisms are more sensitive to the effects of CGA-277476 than are adults.

However, Novartis Crop Protection has determined that when an additional tenfold safety margin is used, the percent of the RfD that will be utilized by aggregate exposure to residues of CGA-277476 is 0.8 percent for nursing infants less than 1 year old, 3.5 percent for non-nursing infants, 1.4 percent for children 1 to 6 years old and 1.1 percent for children 7 to 12 years old. 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 to infants and children will result from aggregate exposure to CGA-277476 residues.

3. Siemer and Associates

PP 6F4789

EPA has received a pesticide petition (PP 6F4789) from Siemer & Associates, Inc. on behalf of National Chelating, 4672 West Jennifer, Suite 103, Fresno, CA 93722, 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 an exemption from the requirements for a tolerance for ammonium thiosulfate when used for blossom thinning on apples.

Pursuant to the section 408(d)(2)(A)(i) of the FFDCA, as amended, Siemer & Associates, Inc. on behalf of National Chelating has submitted the following summary of information, data and arguments in support of their pesticide petition. This summary was prepared by Siemer & Associates, Inc. and EPA has not fully evaluated the merits of the petition. EPA edited the summary to clarify that the conclusions and arguments were the petitioner's and not necessarily EPA's and to remove certain extraneous material.

On August 30, 1996 Siemer & Associates on behalf of National Chelating petitioned the EPA, under pesticide petition 6F4789, for a permanent exemption from the requirements of a tolerance for ammonium thiosulfate on apples.

Section 408(b)(2)(A) of the amended Federal Food, Drug, and Cosmetic Act allows the EPA to establish an exemption from the requirements for a tolerance only if the Administrator determines that there is a ''reasonable certainty that no harm will result from the aggregate exposure to the pesticide chemical residue, including all anticipated dietary exposures and all other exposures for which there is reliable information.''

The available information indicates that there is a reasonable certainty that no harm will result from various types of exposure. Requests for waivers from the requirements of performing studies for known chemistry are presented and

substantiated. The following is a summary of the information submitted to the EPA to support the establishment, under Section 408(b)(2)(D) of the amended FFDCA, of a tolerance for ammonium thiosulfate on apples.

A. Residue Chemistry

1. *Plant metabolism*. The qualitative nature of the residues of ammonium thiosulfate in apple is adequately understood. The requirement for residue studies was waived by EPA based on the knowledge that ammonium thiosulfate has been used as a soil applied and foliar applied fertilizer for many years. Prior experience and numerous publications teach that ammonium thiosulfate ionizes when placed into water, forming an ammonium ion and a thiosulfate ion which further degrades to form elemental sulfur and a sulfate ion. The sulfur is further oxidized to form a sulfate ion. The ammonium and sulfate ions thus formed are absorbed into the growing plant and moved into the naturally occurring nitrogen and sulfate pools that occur naturally in growing plants. Once applied to the plant, without isotope identification, it is not possible to separate the ammonium and sulfate ions that will occur from those that already occur naturally in the plant. On this basis, an exemption from the requirements of a tolerance is justified. There is no analytical method needed since there is no practical way to separate the ammonium and sulfate ions from those that naturally occur.

2. *Analytical method*. The need for an analytical method is waived on the basis that there is no need for analyzing for the component of ammonium and sulfate ion applied for blossom thinning purposes.

3. *Magnitude of residues*. No residues of ammonium thiosulfate will be identified separately from those ammonium and sulfate ions naturally occurring. This result supports the proposed exemption from the requirements for a tolerance.

B. Toxicological Profile

A request to waive the battery of mammalian toxicity studies for ammonium thiosulfate is based on and justified by the following:

1. *Acute toxicity*. Based on EPA criteria, ammonium thiosulfate previously registered for a non-food use as an ornamental herbicide has been shown to be relatively non-toxic and has been registered for non-food use purposes as a Category III herbicide. These data have previously been supplied to the agency.

2. *Genotoxicity*. A request for a waiver from the following requirements is made on the basis that sodium thiosulfate is on the FDA Generally Recognized as Safe (GRAS) list at 21 CFR 184.1807, and ammonium thiosulfate is already exempted from the requirements of a tolerance when used in accordance with good agricultural practices as inert (or occasionally active) ingredients in pesticide formulations applied to growing crops or to raw agricultural commodities after harvest (at 40 CFR 180.1001(c)). Ammonium thiosulfate ionizes to form ammonium ion and thiosulfate ion in water with neither of these ions being mutagenic or genotoxic. On that basis the following tests are requested to be waived.

i. Gene Mutation - Ames.

ii. *In vitro* Structural chromosomal aberration assay.

iii. *In vitro* CHO/HGPRT assay.

iv. *In vivo* micronucleus aberration assay.

3. *Reproductive and developmental toxicity*. A request for waiving the data requirements for the following is made on the basis listed above for ''B''. In addition, all of the tests listed below rely on feeding the test substance, to animals that have acidic stomachs. Placing ammonium thiosulfate into an acidic environment will cause near instantaneous ion formation giving rise to ammonium and thiosulfate ions, which ultimately breaks down to elemental sulfur and sulfite. These sulfur forms will be quickly oxidized under acidic conditions to sulfate, which will be incorporated into the normal sulfate pool that exists within the metabolic system of the various animal test systems. The ammonium ion will react with the acidic component, most likely forming ammonium chloride which will be metabolized in a well understood pathway in the systems of the various animal test systems. The new moiety formed in this acidic medium is the sulfite ion which also is well understood and is quickly oxidized to sulfate. The FDA instituted studies in 1975 and 1985 on the GRAS status of sulfite and, as a result of these studies, has substantiated the GRAS status except for a few individuals that might be allergic to sulfite. In this proposed usage however, the sulfite will not reach the possibly allergic people, since the sulfite will be metabolized to sulfate in the plant system before reaching any sensitive people who may consume the treated tissue. The data waivers requested are as follows:

i. Teratology in rats.

ii. Teratology in rabbits.

iii. 2–Generation reproduction in rats.

4. *Subchronic Toxicity*. The data requirements listed below are requested to be waived on the basis illustrated above at paragraph 3.

i. 28–Day dermal in rats.

ii. 13–Week oral feeding in rats.

iii. 90–Day oral feeding in dogs.

5. *Chronic toxicity*. The data requirements listed below are requested to be waived for reasons listed above at paragraph 3.

i. 1–Year chronic toxicity in dogs.

ii. 18–Month chronic toxicity & carcinogenicity in mice.

iii. 24–Month chronic toxicity & carcinogenicity in rats.

6. *Animal metabolism*. The metabolism of ammonium thiosulfate is well understood in animals. As listed above, this substance rapidly ionizes in the acidic portion of the animal gut, giving rise to ammonium ion and sulfate ion. Both of these substances are required and occur in the metabolism of animals.

7. *Metabolite toxicology*. No toxicologically significant metabolites will be detected in plant or animal metabolism studies using ammonium thiosulfate. Therefore, no metabolites are required to be regulated.

8. *Endocrine effects*. There is no information available that suggest that ammonium thiosulfate would be associated with endocrine effects.

C. Aggregate Exposure

1. *Dietary exposure—i. Food*. There will be no residues of ammonium thiosulfate that will reach any portion of the US population as a result of using ammonium thiosulfate as a blossom thinner on apples. The ammonium and sulfate ions that will arise will not be different from the naturally occurring forms of the ions, which exceed by far the amount that will be applied as a result of the use of the ammonium thiosulfate.

ii. *Drinking water*. Ammonium and sulfate ions that arise from ammonium thiosulfate use will add no additional burden to the drinking water. The end points of the two ions formed as a result of ammonium thiosulfate use will both be used in plant nutrition. The ammonium form of nitrogen resists leaching by binding to the colloid fraction in the soil to resist ground water contamination. The amount of sulfate added as a result of the described use will add an imperceptible amount to the sulfate level already in existence in the soil.

There is a reasonable certainty that no harm will result from dietary exposure to ammonium thiosulfate, because dietary exposures to residues on food cannot be differentiated from those that

will occur naturally in food, and exposure through drinking water is expected to be insignificant.

2. *Non-dietary exposure*. There is no non-dietary exposure expected, since any ammonium thiosulfate finding its way onto the plants or around any plants will be absorbed and metabolized into naturally occurring plant constituents.

D. Cumulative Effects

There are no cumulative effects expected since the ammonium thiosulfate metabolites are all incorporated into naturally occurring constituents found in all plant systems.

E. Safety Determination

1. *U.S. population*. The natural occurrence of the metabolites of the ammonium and sulfate ions in all plants and in humans is the basis for the Generally Recognized As Safe characterization of the thiosulfate ion and the use of the ammonium ion as a component in nearly all fertilizers, supports the conclusion that there is a ''reasonable certainty of no harm'' from aggregate exposure to ammonium thiosulfate.

2. *Infants and children*. No developmental, reproductive or fetotoxic effects have been associated with ammonium thiosulfate and its use as a fertilizer. The calculation of safety margins with respect to ammonium thiosulfate is unnecessary since the ammonium and sulfate ions that will arise from the use of ammonium thiosulfate will add only slightly to the already naturally occurring nitrogen and sulfur pools in existence in various plants. Since there will be no residues of toxicological significance resulting from ammonium thiosulfate, calculations of safety margins are not necessary based on the lack of any unnatural residues.

F. International Tolerances

There is no Codex maximum residue level established for ammonium thiosulfate on apple. However, ammonium thiosulfate is widely used as a nutrient in many parts of the world.

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BILLING CODE 6560–50–F

ENVIRONMENTAL PROTECTION AGENCY

[OPP–181046; FRL 5717–1]

Carbofuran; Receipt of Application for Emergency Exemption, Solicitation of Public Comment

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice.

SUMMARY: EPA has received a specific exemption request from the Arkansas State Plant Board (hereafter referred to as the ''Applicant'') to use the pesticide flowable Carbofuran (Furadan 4F Insecticide/Nematicide) (EPA Reg. No. 279–2876) to treat up to 1 million acres of cotton to control cotton aphids. The Applicant proposes the use of a chemical which has been the subject of a Special Review within EPA's Office of Pesticide Programs. The granular formulation of carbofuran was the subject of a Special Review between the years of 1986 – 1991, which resulted in a negotiated settlement whereby most of the registered uses of granular carbofuran were phased out. While the flowable formulation of carbofuran is not the subject of a Special Review, EPA believes that the proposed use of flowable carbofuran on cotton could pose a risk similar to the risk assessed by EPA under the Special Review of granular carbofuran. Therefore, in accordance with 40 CFR 166.24, EPA is soliciting public comment before making the decision whether or not to grant the exemption.

DATES: Comments must be received on or before May 28, 1997.

ADDRESSES: Three copies of written comments, bearing the identification notation ''OPP–181046,'' should be submitted by mail to: Public Information and Records Integrity Branch, Information Resources and Services Division (7506C), Office of Pesticide Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460. In person, bring comments to: Rm. 1132, Crystal Mall #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 (CBI) should be submitted through e-mail.

Information submitted in any comment concerning this notice may be claimed confidential 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. A copy of the comment that does not contain CBI must be provided by the submitter for inclusion in the public record. Information not marked confidential may be disclosed publicly by EPA without prior notice. All written comments filed pursuant to this notice will be available for public inspection in Rm. 1132, Crystal Mall No. 2, 1921 Jefferson Davis Highway, Arlington, VA, from 8:30 a.m. to 4 p.m., Monday through Friday, except legal holidays.

FOR FURTHER INFORMATION CONTACT: By mail: David Deegan, Registration Division (7505W), Office of Pesticide Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460. Office location, telephone number and e-mail: Floor 6, Crystal Station #1, 2800 Jefferson Davis Highway, Arlington, VA, (703) 308–8327; e-mail: deegan.dave@epamail.epa.gov.

SUPPLEMENTARY INFORMATION: Pursuant to section 18 of the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA) (7 U.S.C. 136p), the Administrator may, at her discretion, exempt a state agency from any registration provision of FIFRA if she determines that emergency conditions exist which require such exemption. The Applicant has requested the Administrator to issue a specific exemption for the use of carbofuran on cotton to control aphids. Information in accordance with 40 CFR part 166 was submitted as part of this request.

As part of this request, the Applicant asserts that the state of Arkansas is likely to experience a non-routine infestation of aphids during the 1997 cotton growing season. The applicant further claims that, without a specific exemption of FIFRA for the use of flowable carbofuran on cotton to control cotton aphids, cotton growers in much of the state will suffer significant economic losses. The applicant also details a use program designed to minimize risks to pesticide handlers and applicators, non-target organisms (both Federally-listed endangered species, and non-listed species), and to reduce the possibility of drift and runoff.

The applicant proposes to make no more than two applications at the rate of 0.25 lb. active ingredient [(a.i.)], (8 fluid oz.) in a minimum of 2 gallons of finished spray per acre by air, or 10 gallons of finished spray per acre by ground application. The total maximum proposed use during the 1996 growing season (June 1, 1997 until September 30, 1997) would be 0.5 lb. a.i. (16 fluid oz.) per acre. The applicant proposes that