ENVIRONMENTAL PROTECTION AGENCY

[OPP–2004–0363; FRL–7686–5]

Pinoxaden; 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 identification (ID) number OPP–2004–0363, must be received on or before December 20, 2004.

ADDRESSES: Comments may be submitted electronically, by mail, or through hand delivery/courier. Follow the detailed instructions as provided in Unit I. of the **SUPPLEMENTARY INFORMATION**.

FOR FURTHER INFORMATION CONTACT: Jim Tompkins, Registration Division (7505C), Office of Pesticide Programs, Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460–0001; 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 potentially affected by this action if you are an agricultural producer, food manufacturer, or pesticide manufacturer. Potentially affected entities may include, but are not limited to:

- Crop production (NAICS 111)
- Animal production (NAICS 112)
- Food manufacturing (NAICS 311)
- Pesticide manufacturing (NAICS 32532)

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 this unit could also be affected. The North American Industrial Classification System (NAICS) codes have been provided to assist you and others in determining whether this action might apply to certain entities. If you have any 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 Copies of this Document and Other Related Information?

1. *Docket*. EPA has established an official public docket for this action under docket ID number OPP–2004–0363. The official public docket consists of the documents specifically referenced in this action, any public comments received, and other information related to this action. Although a part of the official docket, the public docket does not include Confidential Business Information (CBI) or other information whose disclosure is restricted by statute. The official public docket is the collection of materials that is available for public viewing at the Public Information and Records Integrity Branch (PIRIB), Rm. 119, Crystal Mall #2, 1801 S. Bell St., Arlington, VA. This docket facility is open from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays. The docket telephone number is (703) 305–5805.

2. *Electronic access*. You may access this **Federal Register** document electronically through the EPA Internet under the ''**Federal Register**'' listings at *http://www.epa.gov/fedrgstr/*.

An electronic version of the public docket is available through EPA's electronic public docket and comment system, EPA Dockets. You may use EPA Dockets at *http://www.epa.gov/edocket/* to submit or view public comments, access the index listing of the contents of the official public docket, and to access those documents in the public docket that are available electronically. Although not all docket materials may be available electronically, you may still access any of the publicly available docket materials through the docket facility identified in Unit I.B.1. Once in the system, select ''search,'' then key in the appropriate docket ID number.

Certain types of information will not be placed in the EPA Dockets. Information claimed as CBI and other information whose disclosure is restricted by statute, which is not included in the official public docket, will not be available for public viewing in EPA's electronic public docket. EPA's policy is that copyrighted material will not be placed in EPA's electronic public docket but will be available only in printed, paper form in the official public docket. To the extent feasible, publicly available docket materials will be made available in EPA's electronic public docket. When a document is selected from the index list in EPA Dockets, the system will identify whether the document is available for viewing in EPA's electronic public docket. Although not all docket materials may be available electronically, you may still access any of the publicly available docket materials through the docket facility identified in Unit I.B. EPA intends to work towards providing electronic access to all of the publicly available docket materials through EPA's electronic public docket.

For public commenters, it is important to note that EPA's policy is that public comments, whether submitted electronically or in paper, will be made available for public viewing in EPA's electronic public docket as EPA receives them and without change, unless the comment contains copyrighted material, CBI, or other information whose disclosure is restricted by statute. When EPA identifies a comment containing copyrighted material, EPA will provide a reference to that material in the version of the comment that is placed in EPA's electronic public docket. The entire printed comment, including the copyrighted material, will be available in the public docket.

Public comments submitted on computer disks that are mailed or delivered to the docket will be transferred to EPA's electronic public docket. Public comments that are mailed or delivered to the docket will be scanned and placed in EPA's electronic public docket. Where practical, physical objects will be photographed, and the photograph will be placed in EPA's electronic public docket along with a brief description written by the docket staff.

C. How and To Whom Do I Submit Comments?

You may submit comments electronically, by mail, or through handdelivery/courier. To ensure proper receipt by EPA, identify the appropriate docket ID number in the subject line on the first page of your comment. Please ensure that your comments are submitted within the specified comment period. Comments received after the close of the comment period will be marked ''late.'' EPA is not required to consider these late comments. If you wish to submit CBI or information that is otherwise protected by statute, please follow the instructions in Unit I.D. Do not use EPA Dockets or e-mail to submit CBI or information protected by statute.

1. *Electronically*. If you submit an electronic comment as prescribed in this unit, EPA recommends that you include your name, mailing address, and an e-mail address or other contact information in the body of your comment. Also include this contact information on the outside of any disk or CD ROM you submit, and in any

cover letter accompanying the disk or CD ROM. This ensures that you can be identified as the submitter of the comment and allows EPA to contact you in case EPA cannot read your comment due totechnical difficulties or needs further information on the substance of your comment. EPA's policy is that EPA will not edit your comment, and any identifying or contact information provided in the body of a comment will be included as part of the comment that is placed in the official public docket, and made available in EPA's electronic public docket. If EPA cannot read your comment due to technical difficulties and cannot contact you for clarification, EPA may not be able to consider your comment.

i. *EPA Dockets*. Your use of EPA's electronic public docket to submit comments to EPA electronically is EPA's preferred method for receiving comments. Go directly to EPA Dockets at *http://www.epa.gov/edocket/*, and follow the online instructions for submitting comments. Once in the system, select ''search,'' and then key in docket ID number OPP–2004–0363. The system is an ''anonymous access'' system, which means EPA will not know your identity, e-mail address, or other contact information unless you provide it in the body of your comment.

ii. *E-mail*. Comments may be sent by e-mail to opp-docket@epa.gov, Attention: Docket ID Number OPP–2004–0363. Incontrast to EPA's electronic public docket, EPA's e-mail system is not an ''anonymous access'' system. If you send an e-mail comment directly to the docket without going through EPA's electronic public docket, EPA's e-mail system automatically captures your e-mail address. E-mail addresses that are automatically captured by EPA's e-mail system are included as part of the comment that is placed in the official public docket, and made available in EPA's electronic public docket.

iii. *Disk or CD ROM*. You may submit comments on a diskor CD ROM that you mail to the mailing address identified in Unit I.C.2. These electronic submissions will be accepted in WordPerfect or ASCII file format. Avoid the use of special characters and any form of encryption.

2. *By mail*. Send your comments to: Public Information and Records Integrity Branch (PIRIB) (7502C), Office of Pesticide Programs (OPP), Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460–0001, Attention: Docket ID Number OPP–2004–0363.

3. *By hand delivery or courier*. Deliver your comments to: Public Information and Records Integrity Branch (PIRIB), Office of Pesticide Programs (OPP), Environmental Protection Agency, Rm. 119, Crystal Mall #2, 1801 S. Bell St., Arlington, VA, Attention: Docket ID Number OPP–2004–0363. Such deliveries are only accepted during the docket's normal hours of operation as identified in Unit I.B.1.

D. How Should I Submit CBI to the Agency?

Do not submit information that you consider to be CBI electronically through EPA's electronic public docket or by e-mail. You may claim information that you submit to EPA as CBI by marking any part or all of that information as CBI (if you submit CBI on disk or CD ROM, mark the outside of the disk or CD ROM as CBI and then identify electronically within the disk or CD ROM the specific information that is 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 docket and EPA's electronic public docket. If you submit the copy that does not contain CBI on disk or CD ROM, mark the outside of the disk or CD ROM clearly that it does not contain CBI. Information not marked as CBI will be included in the public docket and EPA's electronic public docket without prior notice. If you have any questions about CBI or the procedures for claiming CBI, please consult the person listed 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 ID 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 Cosmetic Act (FFDCA), 21 U.S.C. 346a. EPA has determined that this petition contains data or information regarding theelements set forth in FFDCA section 408(d)(2); however, EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data supports granting of the petition. Additional data may be needed before EPA rules on the petition.

List of Subjects

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

Dated: November 3, 2004

Lois Rossi,

Director, Registration Division, Office of PesticidePrograms.

Summary of Petition

The petitioner summary of the pesticide petition is printed below as required by FFDCA section 408(d)(3). The summary of the petition was prepared by the petitioner and represents the view of the petitioner. 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.

Syngenta Crop Protection

EPA has received a pesticide petition 4F6817 from Syngenta Crop Protection, Inc., P.O. Box 18300, Greensboro, North Carolina, 27419–8300 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 establishing a tolerance for residues of pinoxaden in or on the raw agricultural commodities (RAC) wheat grain at 0.70 parts per million (ppm), wheat, forage at 3.0 ppm, wheat, hay at 1.75 ppm, wheat, straw at 1.5 ppm, barley, grain at 0.70 ppm, barley, hay at 1.25 ppm, and barley, straw at 0.60 ppm. 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*. Metabolism of pinoxaden was studied in wheat using radiolabeled pinoxaden. The metabolism in plants is well understood and the data is adequate for selection of residues of concern for tolerance setting purposes. The metabolic profile in plants supports the use of an analytical method that accounts for parent pinoxaden and its major metabolites.

2. *Analytical method*. Syngenta Crop Protection, Inc., has submitted practical analytical methodology for detecting and measuring levels of pinoxaden and its three major metabolites. The method is based upon commodity specific cleanup procedures and High Performance Liquid Chromatography (HPLC) determination with triple stage quadruple mass spectrometry (LC/MS/MS). The limit of quantitation (LOQ), as demonstrated by the lowest acceptable recovery samples, is 0.01 ppm for grain, and 0.02 ppm for forage, hay, and straw.

3. *Magnitude of residues*. A magnitude of the residueprogram was performed with pinoxaden on full guideline geography to support uses on all types of wheat, and barley crops.

B. Toxicological Profile

1. *Acute toxicity*. Pinoxaden technical and the end-use formulation have very low acute toxicity by oral, dermal, and inhalation exposure routes. For pinoxaden technical, the oral LD_{50} in rats is >5,000 millgrams/kilogram (mg/kg). The rat dermal LD_{50} is >2,000 mg/kg and the rat inhalation LC_{50} is 5.22 milligrams/liter (mg/L) air. Pinoxaden technical is irritating to the eye and non-irritating to the skin. The end-use formulation is mildly to moderately irritating to the eye and skin, the oral LD_{50} in rats is 3,129 mg/kg, the rat dermal LD_{50} is >2,000 mg/kg and the rat inhalation LC_{50} is >5 mg/L. Neither the technical nor the formulation are skin sensitizers.

2. *Genotoxicty*. Pinoxaden has been tested for its potential to induce gene mutation and chromosomal changes in six different test systems. Pinoxaden technical was negative in a bacterial gene mutation assay, a mouse lymphoma mammalian cell mutation assay and an unscheduled DNA synthesis (UDS) assay in rat hepatocytes. In *in vitro* tests for chromosome aberrations in Chinese hamster ovary cells, a small dose related increase was observed at dose levels that produced cytotoxicity. To assess the biological significance of this single positive *in vitro* finding, two in *vivo* tests were performed. When tested in a micronucleus test in bone marrow cells of the mouse at dose levels up to a limit dose of 2,000 mg/kg, pinoxaden did not induce micronuclei, and produced no significant toxicity in the animals. In an *in vivo* UDS study in rats, pinoxaden was negative in this assay for DNA repair. Based on the complete database, it is concluded that pinoxaden is not genotoxic.

3. *Reproductive and developmental toxicity*. Pinoxadenproduced no evidence of reproductive toxicity. In a rat multi-generation reproduction study, pinoxaden technical was administered orally by gavage to rats at dosages of 0, 10, 50, 250, and 500 mg/kg/day over two generations. At 500 mg/kg, parental toxicity was observed as decreased body weight gain (F_0 males) and kidney pathology accompanied by increased water consumption (F_0 and F_1 males and females.) At 500 mg/kg/day, F_1 and F_2 pups had lower body weight gain during lactation. Changes in organ weights were seen in pups at this dose level, but no treatment-related adverse findings were observed for pups ineither generation upon histologic examination. At 10, 50 and 250 mg/kg/day, there was no indication of any adverse effects of treatment. On the basis of the results obtained in this study, the no observed adverse effect level (NOAEL) for both sexes and generations was 250 mg/kg/day. There were no effects on the reproductive parameters and the NOAEL for reproductive toxicity was >500 mg/kg/day. Offspring effects were minor and were observed only at dose levels that produced parental toxicity. There were no indications of any differences in sensitivity to pinoxaden exposure between the different generations or between parental animals andoffspring, and it is concluded that pinoxaden does not cause reproductive toxicity.

In a rat teratogenicity study, pinoxaden technical was administered by gavage to 24 pregnant rats per group at dose levels of 0, 3, 30, 300 or 800 mg/kg/day from days 6 through 20 of gestation. Maternal body weight gain was significantly reduced at the top two dose levels compared to controls. There was no effect of treatment on the number of implantation sites, post-implantation loss, live litter size, and sex ratios, and no significant findings were observed in the maternal animals upon necropsy. Gravid uterus weights, carcass weights and net weight change from day 6 post coitum were significantly reduced at the top dose level. In the presence of the maternal toxicity, mean fetal body weights were reduced at 800 mg/kg/day, and slightly reduced ossification was observed at both 800 and 300 mg/kg bw/day. There were no treatment-related external or visceral observations in the fetuses. Pinoxaden, was not teratogenic in rats when tested under the conditions of this study. The no observed effect level (NOEL) for both maternal and developmental toxicity was 30 mg/kg/day.

Pinoxaden, was evaluated in rabbit developmental toxicity studies. In an initial guideline rabbit study, pinoxaden technical was administered by gavage to pregnant rabbits at dose levels of 0, 10, 30, and 100 mg/kg/day from days 7 through 28 of gestation. Maternal body weight gain was significantly reduced at 100 mg/kg/day. Fetal body weight was reduced at the 100 mg/kg dose level. A second guideline developmental toxicity study was conducted in the rabbit at 0, 10, 30, and 100 mg/kg/day. Maternal toxicity was observed at 30 and at 100 mg/kg/day in the form of reduced overall weight gain compared to control animals. There was no effect of treatment on the number, growth or survival of the fetuses *in utero* and no evidence for an adverse effect on fetal development. There were no treatment-related fetal external, visceral or skeletal findings. In conclusion, the full set of studies indicated that pinoxaden is not teratogenic in rabbits. The maternal NOEL was 10 mg/kg/day, and the NOEL for developmental toxicity was 30 mg/kg/day.

In conclusion, there is no evidence that developing offspring are more sensitive than adults to the effects of pinoxaden, and it is concluded, that pinoxaden does not cause primary developmental toxicity or reproductive toxicity.

4. *Subchronic toxicity*. Pinoxaden technical was evaluated in a number of subchronic studies. In a 3–month gavage study in rats the NOAEL was 300 mg/kg, the highest dose tested. Higher doses in a 28–day rat study caused kidney toxicity at 600 mg/kg, with a NOAEL of 300 mg/kg. In a 3–month gavage study in mice, the NOAEL was 100 mg/kg. Effects at higher dose levels involved reduced body weights at 1,000 mg/kg, reduced hemoglobin at doses greater than or equal to 400 mg/kg (females only) and renal tubule basophilia and increased water consumption in males at 1,000 mg/kg. In a 3–month study in dogs the NOAEL was 100 mg/kg, and inappetance, body weight loss and gastro-intestinal effects were seen at 250 mg/kg. In a 28–day dermal (rat) study,

the NOAEL was 1,000 mg/kg, the highest dose tested, and only a mild, low-grade inflammatory response at the treatment site was noted. In a 90–day subchronic neurotoxicity study in rats, pinoxaden was not neurotoxic when administered by gavage at dose levels of 0, 10, 100 and 500 mg/kg/day. There were no treatment-related neurobehavioral or motor activity effects, no macroscopic findings and no microscopic findings in central or peripheral nervous tissue. In addition, pinoxaden was devoid of any acute neurotoxic effects when administered to rats at a single oral dose of up to 2,000 mg/kg.

5. *Chronic toxicity*. Pinoxaden was not oncogenic in rats or mice. In a 2–year combined carcinogenicity/chronic toxicity study in rats, pinoxaden technical was administered by daily gavage at dose levels of 0, 1, 10, 100, 250, and 500 mg/kg/day. Toxicity was observed in the form of decreased body weight (500 and 250 mg/kg/day), depressed survival (500 and 250 mg/kg/day males only), and kidney pathological changes (500, 250, and 100 mg/kg/day). The kidney pathology was associated with changes in blood chemistry parameters and other associated effects. A minor and sporadic epithelial thickening of the duodenum was observed mainly at 250 and 500 mg/kg. There was no evidence of a carcinogenic effect in this study. In conclusion, chronic treatment of pinoxaden to rats produced effects in only one major target organ at high dose levels, involving chronic progressive nephropathy and associated effects related to kidney toxicity. The NOEL was 10 mg/kg for males and females based on kidney effects at 100 mg/kg and above, and pinoxaden was not carcinogenic.

In an 18–month mouse oncogenicity study, pinoxaden technical was administered by gavage at dose levels of 0, 5, 40, 300 and 750 mg/kg body. Toxicity was observed in the form of decreased body weight gain at 300 mg/kg/day (females only) and 750 mg/kg/day (males and females), decreased survival rates (40, 300 and 750 mg/kg/day males), minor hematology effects (300 and 750 mg/kg), increased liver weights (300 and 750 mg/kg, with increased glycogen deposition) and increased kidney weights (750 mg/kg in females only). Increased epithelial thickening occurred in the small intestine of males and females at 300 and 750 mg/kg. The reduced survival at the higher dose levels in males was a consequence of the gavage dosing procedure, as demonstrated by macro- and micropathology evidence of lung involvement as the single major factor contributing to death. Other than increased mortality in males, there were no treatment-related effects at 40 mg/kg/day. The NOEL for this study was 5 mg/kg for both males and females, and pinoxaden was not carcinogenic.

In a 12–month chronic oral toxicity study in dogs, pinoxaden technical was administered by capsule at dose levels of 0, 5, 25 or 125 mg/kg/day. At 25 and 125 mg/kg/day, treatment-related clinical observations were limited to an increased incidence of salivation at dosing and minor gastrointestinal effects, which were not considered adverse. There were noadverse effects on body weights or food consumption. Minor changes in hematology and blood clinical chemistry parameters were observed at 25 and 125 mg/kg/day compared to control animals. However, due to the small magnitude of the effects and the absence of any treatment-related effects on organ weights or any pathology findings, these clinical pathology changes are considered to be of no toxicological significance. There were no treatment-related micropathology changes seen at any dose level. The NOAEL in this study was 125 mg/kg/day.

6. *Animal metabolism*. Animal metabolism of pinoxaden is well understood. Pinoxaden is rapidly absorbed and excreted when administered to rats, and tissue residues are extremely low, with no accumulation upon repeated dosing. Similar rapid absorption and excretion was seen in mice and rabbits. The metabolic pathway is similar in rodents, rabbits, goats and hens.

7. *Metabolite toxicology*. Toxicity of pinoxaden metabolites has been tested and is well understood. The toxicological profile of all metabolites supports the proposed definition of residue.

8. *Endocrine disruption*. Pinoxaden does not belong to a class of chemicals known or suspected of having adverse effects on the endocrine system. There is no evidence that pinoxaden has any effect on endocrine function in developmental or reproductive studies. Furthermore, histological investigation of endocrine organs in chronic dog, mouse, and rat studies did not indicate that the endocrine system is targeted by pinoxaden.

C. Aggregate Exposure

1. *Dietary exposure*. The potential for chronic and acute dietary exposure from pinoxaden through food and water sources is addressed below.

i. *Food*. Dietary (food) risk evaluations for pinoxaden were performed using field trial residues. A percent of crop treated value of 20% was estimated for wheat and barley based upon Syngenta's estimates of economic, pest, and competitive pressures. Wheat and barley are the only RAC included in the assessment. For the chronic assessments, the average wheat and barley field trial residue values were utilized. For the acute assessment, the two highest field trial residues were averaged and this highest average field trial residue (HAFT) was used in the assessment for all non-blended and partially blended commodities. All dietary exposure evaluations were made using the Dietary Evaluation Model (DEEM™, version 7.87) from Exponent, Inc. and the USDA's Continuing Survey of Food Intake by Individuals (1994–96) with the supplemental 1998 children's survey. Chronic exposure was compared to a chronic reference dose of 0.10 mg/kg bw/day is based upon a NOAEL of 10 mg/kg bw/day from the chronic rat study. The acute reference dose of 0.3 mg/kg bw/day for the subpopulation of women 13–49 years of age is based upon the developmental NOAEL of 30 mg/kg/day in developmental toxicity studies in rabbits. For all other subpopulations, the acute reference dose of 1.5 mg/kg bw/day is based upon a NOAEL for acute effects at 150 mg/kg/day in a range finding rabbit developmental toxicity study. A 100x uncertainty factor was assumed for both the chronic and acute assessments. The chronic exposures were expressed as a percent of a reference dose of 0.10 mg/kg bw/day. The acute exposures (at the 99.9th percentile) were expressed as a percent of a reference dose of 0.3 mg/kg bw/day for women 13–49 years of age and 1.5 mg/kg bw/day for all other subpopulations. Secondary residues in animal commodities were calculated by constructing diets for beef and dairy cattle, poultry and swine in order to calculate anticipated residues in meat, fat, milk and pork. The beef cattle diet was used to calculate meat, fat and organ meats residues. The dairy cattle diet was used to estimate residues in milk. The swine diet was used for secondary residues in pork commodities and the poultry diet was used for residues in poultry commodities. The chronic animal diet was calculated using averaged field trial residues where as the acute animal diet used averaged field trial residues on blended commodities such as grain and the HAFT on non-blended commodities such as hay, straw and forage. Beef (cattle and dairy) and swine transfer factors were derived from a lactating goat metabolism study, where the

animals were dosed with 121 ppm pinoxaden. Poultry transfer factors were derived from a hen metabolism study, where the animals were dosed with 97 ppm pinoxaden.

The results were favorable in both acute and chronic assessment scenarios. Acute exposures at the (99.9th percentile) were 0.11% of the acute reference dose (0.3 mg/kg bw/day) for women 13–49 years of age, and less than 0.05% for all other subpopulations. The chronic exposure values were negligible (<0.05% of the chronic reference dose of 0.10 mg/kg bw/day for all subpopulations).

ii. *Drinking water*. The acute estimated environmental concentrations of pinoxaden (including the major degradates) in surface and ground water are 1.366 ppb (PRZM/EXAMS) and 0.003234 ppb (SCI-GROW), respectively. The acute Population Adjusted Dose (aPAD) for pinoxaden (plus degradates) is 0.3 mg/kg bw/day for women 13–49 years of age and 1.5 mg/kg bw/day for all other population subgroups. From the acute dietary exposure analysis, the highest acute food exposure from the uses of pinoxaden was 0.000509 mg/kg/day at the 99.9th percentile for the 20–49 years old subpopulation. Using this information, acute drinking water levels of comparison (DWLOC acute) were calculated for pinoxaden and the major degradates, ranging from 8,990 to 52,487 ppb. Based on this analysis, pinoxaden (plus degradates) estimated environmental concentrations (EECs) do not exceed the calculated acute DWLOCs. The chronic estimated environmental concentration of pinoxaden (including the major degradates) in surface water is 0.21137 ppb (annual average value from PRZM/EXAMS). The chronic PAD for pinoxaden (plus degradates) is 0.10 mg/kg bw/day. From the chronic dietary exposure analysis, the highest exposure estimate of 0.000047 mg/kg bw/day was determined for the children 1–2 years old subpopulation. Based on the EPA's ''Interim Guidance for Conducting Drinking Water Exposure and Risk Assessments'' document (62 FR 63662, December, 2, 1997), chronic DWLOC chronic were calculated for pinoxaden (plus degradates), ranging from 999.5 to 2999.4 ppb. Based on this analysis, pinoxaden (plus degradates) EECs do not exceed the calculated chronic DWLOCs.

2. *Non-dietary exposure*. There are no sources ofnon-dietary exposure, as pinoxaden will be registered for agricultural uses only and will not be available for any residential or public uses.

D. Cumulative Effects

The potential for cumulative effects of pinoxaden and other substances that have a common mechanism of toxicity has also been considered. Pinoxaden, is a member of the new phenylpyrazolin class of herbicides. There is no reliable information to indicate that toxic effects produced by pinoxaden would be cumulative with those of any other chemical including another pesticide. Therefore, Syngenta believes it is appropriate toconsider only the potential risks of pinoxaden in an aggregate risk assessment.

E. Safety Determination

1. *Infants and children*. In assessing the potential for additional sensitivity of infants and children to residues of pinoxaden, data from developmental toxicity studies in the rat and rabbit and a two-generation reproduction study in the rat have been considered. In a multi-generation reproductive study, there were no indications of any differences in sensitivity to pinoxaden exposure between the different generations or between animals and offspring. The parental NOAEL for both sexes was considered to be 250 mg/kg/day. Offspring effects were not observed at dose levels that did not produce parental toxicity. Pinoxaden was not teratogenic and not directly toxic to the progeny in a developmental toxicity study in rats. The NOEL for both maternal and developmental toxicity was 30 mg/kg/day. Pinoxaden was not teratogenic in rabbits, and the maternal NOEL was 10 mg/kg/day. The NOEL for fetuses was 30 mg/kg/day. Since the NOEL for fetal effects was higher than the NOEL for maternal effects, there was no indication of a greater sensitivity of fetuses to pinoxaden administration. 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 prenatal and postnatal toxicity and the completeness of the database. Based on the current toxicological requirements, the database for pinoxaden relative to prenatal and postnatal effects for children is complete. Further, the developmental studies showed no increased sensitivity in fetuses as compared to maternal animals following *in utero* exposures in rats and rabbits, and no increased sensitivity in pups as compared to the adults in the multi-generation reproductive toxicity study. Therefore, it is concluded that an additional uncertainty factor is not warranted to protect the health of infants and children and that RfDs of 0.3 mg/kg/day (acute exposures to women 13–50 yrs of age), 1.5 mg/kg/day (acute exposures to general population) and 0.10 mg/kg/day (chronic expsoures) are appropriate for assessing aggregate risk to infants and children of pinoxaden. Chronic and acute aggregate exposures to all infants (<1 year old) is less than 0.2% of the acute and chronic RfDs. Therefore, based on the completeness and reliability of the toxicity database, Syngenta concludes that there is reasonable certainty that no harm will result to infants and children from aggregate exposure to pinoxaden residues.

F. International Tolerances

There are no tolerances or maximum residue limits set for pinoxaden in any country at the time of this filing.

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BILLING CODE 6560–50–S

ENVIRONMENTAL PROTECTION AGENCY

[FRL–7838–9]

E–Docket ID No. ORD–2004–0003; Draft Proposed Sampling Program To Determine Extent of World Trade Center Impacts to the Indoor Environment

AGENCY: Environmental Protection Agency.

ACTION: Notice of Extension of Public Comment Period for Draft Proposed Sampling Program to Determine Extent of World Trade Center Impacts to the Indoor Environment.

SUMMARY: On October 21, 2004, EPA published a **Federal Register** notice (69 FR 61838) announcing the availability of the External Review Draft entitled, *Draft Proposed Sampling Program to Determine Extent of World Trade Center Impacts to the Indoor Environment* (EPA/600/R–04/169A), and the beginning of a 30-day public comment period. At the request of members of the Lower Manhattan community and labor organizations who have said an extension is needed for them to formulate their comments, EPA is extending the public comment period until January 3, 2005. EPA will consider the public comment submissions in revising the document.

DATES: The public comment period will end on January 3, 2005. Technical comments should be in writing and must be postmarked by January 3, 2005.

ADDRESSES: The External Review Draft, *Draft Proposed Sampling Program to Determine Extent of World Trade Center Impacts to the Indoor Environment,* is