an EPA Pesticide Fact Sheet on cyprodinil *N*-(4-cyclopropyl-6-methyl-pyrimidin-2-yl)-aniline.

A paper copy of this fact sheet, which provides a summary description of the chemical, use patterns and formulations, science findings, and the Agency's regulatory position and rationale, may be obtained from the National Technical Information Service (NTIS), 5285 Port Royal Road, Springfield, VA 22161.

In accordance with section 3(c)(2) of FIFRA, a copy of the approved label, the list of data references, the data and other scientific information used to support registration, except for material specifically protected by section 10 of FIFRA, are available for public inspection in the Public Information and Records Intregrity Branch, Information Resources and Services Division (7502C), Office of Pesticide Programs, Environmental Protection Agency, Rm. 119, CM #2, Arlington, VA 22202 (703–305–5805). Requests for data must be made in accordance with the provisions of the Freedom of Information Act and must be addressed to the Freedom of Information Office (A-101), 401 M St., SW., Washington, D.C. 20460. Such requests should: (1) Identify the product name and registration number and (2) specify the data or information desired.

Authority: 7 U.S.C. 136.

List of Subjects

Environmental protection, Pesticides and pests, Product registration.

Dated: May 27, 1998.

James Jones,

Director, Registration Division, Office of Pesticide Programs.

[FR Doc. 98–15013 Filed 6–4–98; 8:45 am]

BILLING CODE 6560–50–F

ENVIRONMENTAL PROTECTION AGENCY

[PF–807; FRL–5791–4]

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–807, must be received on or before July 6, 1998.

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

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

Information submitted as a comment concerning this document may be claimed confidential by marking any part or all of that information as ''Confidential Business Information'' (CBI). CBI should not be submitted through e-mail. Information marked as CBI will not be disclosed except in accordance with procedures set forth in 40 CFR part 2. A copy of the comment that does not contain CBI must be submitted for inclusion in the public record. Information not marked confidential may be disclosed publicly by EPA without prior notice. All written comments will be available for public inspection in Rm. 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: The product manager listed in the table below:

Product Manager	Office location/telephone number	Address
Joanne Miller (PM 23) ...	Rm. 237, CM #2, 703–305–6224, e-mail:miller.joanne@epamail.epa.gov.	1921 Jefferson Davis Hwy, Arlington, VA
Beth Edwards (PM 3)	Rm. 206, CM #2, 703–305–5400, e-mail: edwards.beth@epamail.epa.gov.	Do.

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 granting of the petition. Additional data may be needed before EPA rules on the petition.

The official record for this notice of filing, as well as the public version, has been established for this notice of filing under docket control number [PF–807] (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 notice may be filed online at many Federal Depository Libraries.

List of Subjects

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

Dated: May 20, 1998.

James Jones,

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. Novartis Crop Protection, Inc.

PP 7F4924

EPA has received a pesticide petition (PP 7F4924) from Novartis Crop Protection, Inc., P.O. Box 18300, Greensboro, NC 27419-8300 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 to establish tolerances for Clodinafop-propargyl, Propanoic acid, 2-[4-[(5-chloro-3-fluoro-2-pyridinyl)oxy]phenoxy]-,2-propynyl ester, in or on the raw agricultural commodities wheat grain at 0.02 and wheat straw at 0.05 parts per million (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*. The metabolism of CGA-184927 in wheat is understood for the purposes of the proposed tolerance. Two studies, one with the racemic mixture of the R (+) and S (-) forms and the other with the pure R (+) form (CGA-184927 pyridyloxy labeled), gave similar results. Metabolism involves hydrolysis of the parent to the resulting acid followed by conjugation, arylhydroxylation at the 6 position of the pyridyl ring followed by sugar conjugation, and cleavage of the pyridinyloxy-phenoxy ether bridge which forms the breakdown products 2-(4-hydroxyphenoxy) propanoic acid and 2-hydroxy-3-fluoro-5-chloropyridine.

2. *Analytical method*. Novartis has submitted practical analytical methods for the determination of CGA-184927 and its major plant metabolite CGA-193469 in wheat raw agricultural commodities (RACs). CGA-184927 is extracted from crops with acetonitrile, cleaned up by solvent partition and solid phase extraction and determined by column switching HPLC with ultraviolet detection. CGA-193469 is extracted from crops with an acetone-buffer (pH=3) solution, cleaned up by solvent partition and solid phase extraction, and determined by HPLC with UV detection. The limits of quantitation (LOQ) for the methods are 0.02 ppm for CGA-184927 in grain and forage, 0.05 ppm for CGA-184927 in straw, and 0.05 ppm for CGA-193469 in forage, straw and grain.

3. *Magnitude of residues*. Twelve residue trials were conducted from 1989-1992 in the major spring wheat growing areas of Manitoba, Alberta and Saskatchewan, which share compatible crop zones with the major spring wheat growing areas of the US (MT, ND, SD, MN). Nine trials were conducted in 1989-91 with a tank mix of CGA-184927 and a safener as separate EC formulations, and three trials in 1992 were conducted with CGA-184927 and the safener as a pre-pack EC formulation. All trials had a single post-emergence application of CGA-184927 at a rate of 80 g a.i./Ha. At PHIs of 66-97-days, no detectable residues of CGA-184927 or its metabolite CGA-193469 were found in mature grain and straw from these trials. Separate decline studies (3) on green forage showed no detectable residues of CGA-184927 or CGA-193469 beyond the 3-days after application (DAA) interval. A freezer storage stability study indicated reasonable stability of both analytes for a period of 1-year, with CGA-184927 showing a decline to 56% in grain and 47% in straw after 2-years. CGA-193469 remained stable for at least 2-years.

B. Toxicological Profile

1. *Acute toxicity*. The acute oral and dermal LD_{50} values for clodinafop-propargyl are 1829 mg/kg and greater than 2,000 mg/kg for rats of both sexes, respectively. Its acute inhalation LC_{50} in the rat is greater than 2.33 mg/liter, the highest attainable concentration. Clodinafop-propargyl is slightly irritating to the eyes, minimally irritating to the skin of rabbits, but was found to be sensitizing to the skin of the guinea pig. This technical would carry the EPA signal word ''Caution''.

2. *Genotoxicty*. The mutagenic potential of clodinafop-propargyl was investigated in 6 independent studies covering different end points in eukaryotes and prokaryotes *in vivo* and *in vitro*. These tests included: Ames reverse mutation with *Salmonella typhimurium* and Chinese hamster V79 cells; chromosomal aberrations using human lymphocytes and the mouse micronucleus test; and DNA repair using rat hepatocytes and human fibroblasts. Clodinafop-propargyl was found to be negative in all these tests and, therefore, is considered devoid of any genotoxic potential at the levels of specific genes, chromosomes or DNA primary structure.

3. *Reproductive and developmental toxicity*. Dietary administration of clodinafop-propargyl over 2-generations at levels as high as 1,000 ppm did not affect mating performance, fertility or litter sizes. The physiological developmental and the survival of the pups during the last week of the lactation period were slightly reduced at levels equal to or greater than 500 ppm during the first generation only. Target organs were liver (adults) and kidney (adults and pups). The treatment had no effect on reproductive organs. The developmental and reproductive NOEL was 50 ppm, corresponding to a mean daily intake of 3.3 milligrams/kilogram (mg/kg) clodinafop-propargyl.

In a developmental toxicity study in rats, the highest dose level of 160 mg/kg resulted in reduced body weight gain of the dams and signs of retarded fetal body weight and incomplete ossification of vertebrae and sternebrae. No teratogenic activity of the test article was detected. The NOEL for dams and fetuses was 40 mg/kg/day.

In a developmental toxicity study in rabbits, mortality was observed in dams at dose levels of 125 and 175 mg/kg. No teratogenic or fetotoxic effects were noted. The maternal NOEL was 25 mg/kg/day and the fetal NOEL was 175 mg/kg/day.

4. *Subchronic toxicity*. A 90-day feeding study in rats at 1,000 ppm resulted in reduced body weight gain, increased liver weights, hematological changes, and increased serum activities of the alkaline phosphatase. Target organs were liver (increased weight), thymus (atrophy) and spleen (reduced weight). The changes were reversible during 4-weeks of recovery. The NOEL was 15 ppm (0.92 mg/kg in males and 0.94 mg/kg in females).

In a 90-day feeding study in mice, 400 ppm resulted in reduced activity, one death, markedly increased activities of aminotransferases, alkaline phosphatase, and albumin concentration, increased liver weights, hepatocellular hypertrophy, and single cell necroses in all mice. Other findings included intrahepatic bile duct proliferation, Kupffer cell hyperplasia and higher incidence of inflammatory cell infiltration. These findings were considered to be secondary to the hepatocyte necrosis. The NOEL of 6 ppm was equivalent to a daily dose of 0.9 mg/kg in males and 1.05 mg/kg in females.

In a 90-day study in beagle dogs, levels of 500 and 1,000 ppm fed over 1-weeks clearly exceeded a maximum tolerated dose and led to mortality and severe toxicity. Effects at 50 and 200 ppm were limited to dermatitis and

clinical chemistry changes, which were generally mild and transient. The NOEL of 10 ppm was equivalent to a mean daily intake of 0.36 mg/kg in males and females.

5. *Chronic toxicity*. In a 12-month feeding study in dogs, 500 ppm resulted in transient dermatitis and reduced body weight gain. Two females were more severely affected and showed inappetence, body weight loss, tremors and severe dermatitis, and necessitated an interruption of the treatment in order to avoid mortality. Histopathology revealed slight hepatocellular hypertrophy in one male and one female. The NOEL of 100 ppm was equivalent to a mean daily intake of 3.38 mg/kg in males and 3.37 mg/kg in female.

Lifetime dietary administration of clodinafop-propargyl to mice resulted in reduced body weights and reduced survival in males treated at 250 ppm. Severe hepatotoxicity was noted at 100 and 250 ppm in both sexes. Based on markedly increased liver weights, enhanced serum activities of hepatic enzymes and hepatocellular necroses, dietary levels of 100 ppm and 250 ppm clearly exceeded maximum tolerated doses in males and females, respectively. The increased incidence of benign liver tumors that occurred in males treated at 250 ppm was, therefore, considered a toxicologically irrelevant response as the livers of these animals were damaged significantly and this finding was not interpretable. The test substance was severely hepatotoxic at 100 and 250 ppm, with males being more sensitive than females. Based on markedly increased liver weights, enhanced serum activities of hepatic enzymes, and hepatocellular necroses, dietary levels of 100 ppm and 250 ppm clearly exceeded maximum tolerated doses in males and females, respectively. The incidence of hepatocellular carcinoma, in these clearly compromised mice, remained within the historical control range, although the incidence was slightly increased in comparison to the concomitant controls. Tumor incidences in females were generally low and well within the range of the historical controls. The NOEL of 10 ppm was equivalent to a mean daily dose of 1.10 mg/kg in males and 1.25 mg/kg in females.

Dietary treatment of rats with concentrations over 2-years resulted in initial inappetence in males and reduced body weight development in both sexes treated at 750 ppm. The main target organ of toxicity was the liver. Changes in plasma protein and lipid levels, strongly enhanced serum activities of liver enzymes, increased liver weights, and severe hepatocellular necroses were observed at dietary doses of 300 and 750 ppm in males and at 750 ppm in females, giving evidence that these dose levels exceeded a maximum tolerated dose (MTD). Top dose group males showed higher incidences of prostate adenoma, while prostate hyperplasia was reduced. The total incidence of proliferative changes in the prostate remained unchanged. Females treated at the same high dose had higher incidences of ovary tubular adenoma. Both tumors also occur spontaneously in the rat strain used. Their slightly enhanced incidences are likely a consequence of the severe disturbance of the general physiological balance due to excessive liver toxicity. There was no progression to a malignant phenotype and the tumors had no influence on survival. In rats, feeding a dose of 750 ppm to males showed higher incidences of prostate adenoma, while prostate hyperplasia was reduced. The total number of tumor-bearing animals showed no dose-related trends. The NOEL of 10 ppm was equivalent to a mean daily dose of 0.32 mg/kg in males and 0.37 mg/kg in females.

6. *Animal metabolism*. In rats, clodinafop-propargyl was rapidly absorbed through the gastrointestinal tract. Absorption through the skin of rats is considerably slower with 15% of a dermally applied dose being absorbed within 8-hours. Single doses were excreted more rapidly by female rats than by males. Most likely due to enzyme induction, differences were much less pronounced after repeated treatment. Both sexes excreted clodinafop-propargyl with urine and feces mainly in the form of its propionic acid derivative, CGA-193469. Simultaneous administration of the safener, cloquintocet-mexyl, did not alter the rate of excretion of clodinafop-propargyl or its metabolite pattern.

7. *Metabolite toxicology*. Clodinafop-propargyl acts as a typical peroxisome proliferator in the rodent liver which is most likely induced by its propionic acid derivative metabolite, CGA-193469. Like other known well-characterized substances with this property, CGA-193469 caused peroxisome proliferation *in vitro* in hepatocytes of the mouse and rat, but not of the Guinea pig, marmoset, or human. There is ample scientific evidence that exposure to peroxisome proliferators represents no risk of tumor development in man. Clodinafop-propargyl is, therefore, not considered to be a carcinogen of relevance to humans.

8. *Endocrine disruption*. No special studies investigating potential estrogenic or endocrine effects of clodinafop-propargyl have been conducted. However, the standard battery of required studies has been completed. These studies include an evaluation of the potential effects on reproduction and development and an evaluation of the pathology of the endocrine organs following repeated or long-term exposure. Although prostate adenomas and ovarian adenomas were observed to be statistically increased in rats at the highest feeding level with clodinafop-propargyl, this feeding level clearly exceeded the MTD and the livers in these rats were severely compromised. Therefore, these findings are considered irrelevant.

C. Aggregate Exposure

1. *Dietary exposure*. For purposes of assessing the potential exposure under the proposed tolerances for clodinafop-propargyl, Novartis has estimated aggregate exposure based on the theoretical maximum residue contribution (TMRC) from the residues of the active ingredient, clodinafop-propargyl, or metabolites thereof. Residues are below the detection limit in wheat grains and other wheat products, including green wheat used for forage. Tolerances in wheat and wheat products are proposed at the detection limit of 0.02 ppm (LOQ) for the parent active ingredient in wheat grain. Although wheat commodities may be fed to poultry or cattle and it is common practice in some areas to graze cattle on green wheat, tolerances in meat or milk are not necessary because forage commodities do not contain detectable amounts of the parent clodinafop-propargyl or its metabolites.

i. *Chronic*. The RfD of 0.0032 mg/ kg/ day is derived from the male rat NOEL of 0.32 mg/ kg/ day. Based on the assumption that 100% of all wheat used for human consumption would contain residues of clodinafop-propargyl and anticipated residues would be at the level of ½ the LOQ, the potential dietary exposure was calculated using the TAS (TAS Exposure Analysis, Technical Assessment Systems Inc., Washington, DC.) exposure program based on the food survey from the year of 1977/1978. Calculations were made for anticipated residues using ½ the LOQ or 0.01 ppm. The proposed tolerance (0.02 ppm) was set at the lowest limit of detection for the active ingredient in wheat commodities (grain) because, with the available methodology, there are no detectable residues of clodinafop-propargyl in wheat or wheat products. Residues in milk, meat and eggs due to the feeding of wheat grain, green wheat or other

feed commodities will not occur and tolerances for milk, meat and eggs are therefore not required. Calculated on the basis of the assumptions above, the chronic dietary exposure of the U.S. population to clodinafop-propargyl would correspond to 0.000014 mg/kg/day or 0.47% of its RfD. The margin of exposure (MOE) against the NOEL in the most sensitive species is 22,857-fold.

Using the same conservative exposure assumptions, the percentage of the RfD that will be utilized is 0.14% for nursing infants less than 1-year old, 0.34% for non-nursing infants, 1.05% for children 1-6 years old and 0.77% for children 7-12 years old. It is concluded that there is a reasonable certainty that no harm will result to infants and children from exposure to residues of clodinafop-propargyl.

ii. *Acute*. Using the same computer software package used for the calculation of chronic dietary exposure, the acute dietary exposure was calculated for the general population and several sub-populations including children and women of child bearing age. The USDA Food Consumption Survey from 1989-1992 was used, however, instead of the 1977/78 survey used for the chronic assessment. Margins of exposure were calculated against the NOEL of 1 mg/kg found in a 90-day dietary toxicity study in rats, which is the lowest NOEL observed in a short term or reproductive toxicity study. NOEL from reproductive or developmental toxicity studies were significantly higher and there was no evidence that clodinafop-propargyl has any potency to affect these endpoints.

The exposure model predicted that 99.9% of the general population will be exposed to less than 0.000105 mg/kg of clodinafop-propargyl per day, which corresponds to a MOE of almost 9,529 when compared to the NOEL of 1 mg/kg. Children 1-6 years constitute the sub-population with the highest predicted exposure. Predicted acute exposure for this subgroup is less than 0.000136 mg/kg/day, corresponding to a MOE of at least 7,362 for 99.9% of the individuals.

2. *Drinking water*. Other potential sources of exposure of the general population to residues of pesticides are residues in drinking water. Although clodinafop-propargyl has a slight to medium leaching potential, the risk of the parent compound to leach to deeper soil layers is negligible under practical conditions in view of the rapid degradation of the product and its low application rate. According to laboratory and field studies there is no risk of ground water contamination with clodinafop-propargyl or its metabolites. Thus, aggregate risk of exposure to clodinafop-propargyl does not include drinking water. Clodinafop-propargyl is not intended for uses other than the agricultural use on wheat. Thus, there is no potential for non-occupational exposure.

The Maximum Contaminant Level Goal (MCLG) calculated for clodinafop-propargyl according to EPA's procedure leads to an exposure value substantially above levels that are likely to be found in the environment under proposed conditions of use.

MCLG = RfD x 20% x 70 kg/2 L

MCLG = 0.0032 mg/kg x 0.2 x 70 kg/2 L

MCLG = 0.0448 ppm = 44.8 ppb.

3. *Non-dietary exposure*. Exposure to clodinafop-propargyl for the mixer/loader/ground boom/aerial applicator was calculated using the Pesticide Handlers Exposure Database (PHED). It was assumed that the product would be applied 10-days per year by ground boom application to a maximum of 300 acres per day by the grower, 450 acres per day by the commercial ground boom applicator and 741 acres per day by the aerial applicator at a maximum use rate of 28 grams active ingredient per acre. For purposes of this assessment, it was assumed that an applicator would be wearing a long-sleeved shirt and long pants and the mixer/loader would, in addition, wear gloves. These assumptions were selected from PHED. Daily doses were calculated for a 70 kg person assuming 100% dermal penetration. The results indicate that large margins of safety exist for the proposed use of clodinafop-propargyl. Based upon the use pattern for clodinafop, the NOEL (50 mg/kg/day) from the 28-day rat dermal study is appropriate for comparison to mixer/loader-applicator exposure. The chronic NOEL of 0.32 mg/kg/day from the 2-year feeding study in rats is used to examine longer term exposures.

For short-term exposure, MOEs for clodinafop ranged from 2.9E+03 for commercial open mixer-loader to 3.1E+04 for commercial groundboom enclosed-cab applicator. For chronic exposure, MOEs ranged from 6.9E+02 for commercial open mixer-loader to 7.4E+03 for commercial groundboom enclosed-cab applicator. Aerial application of clodinafop results in short-term MOEs of 1.8E+03 for the mixer-loader and 2.0E+03 for pilots. Chronic MOEs are 4.2E+02 for the mixer-loader and 4.7E+02 for the pilot.

In reality, the proposed label will require more restrictive personal protective equipment for applicators and other handlers, resulting in additional margins of safety.

D. Cumulative Effects

A cumulative exposure assessment for effects of clodinafop-propargyl and other substances with the same mechanism of action is not appropriate because there is ample evidence to indicate that humans are not sensitive to the effects of clodinafop-propargyl and other peroxisome proliferators. Thus, the calculations outlined below were done for clodinafop-propargyl alone.

E. Safety Determination

1. *U.S. population*. Using the same conservative exposure assumptions described above, based on the completeness and reliability of the toxicity data, Novartis calculated that the aggregate risk for clodinafop-propargyl for chronic dietary exposure of the U.S. population would correspond to 0.000014 mg/kg/day or 0.47% of its RfD. The margin of exposure (MOE) against the NOEL in the most sensitive species is 22,857-fold. EPA generally has no concern for exposures below 100% of the RfD because the RfD represents the level at or below which daily aggregate dietary exposure over a lifetime will not pose appreciable risks to human health. Therefore, it is concluded that there is a reasonable certainty that no harm will result from aggregate exposure to residues of clodinafop-propargyl.

2. *Infants and children*. In assessing the potential for additional sensitivity of infants and children to residues of clodinafop-propargyl, data from developmental toxicity studies in the rat and rabbit and a 2-generation reproduction study in the rat have been considered. 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.

Retarded fetal body weight and incomplete ossification of vertebrae and sternebrae were observed at a maternally toxic dose of 160 mg/kg/day in rats; however, no teratogenic activity of the test article was detected. The NOEL for dams and fetuses was 40 mg/kg/day. Although mortality was observed in rabbit dams at dose levels of 125 and 175 mg/kg, no teratogenic or fetotoxic effects were noted. The maternal NOEL was 25 mg/kg/day and the fetal NOEL was 175 mg/kg/day.

Clodinafop-propargyl fed over 2-generations to rats at levels as high as 1,000 ppm did not affect mating

performance, fertility, or litter sizes. Physiological developmental and the survival of the pups during the last week of the lactation period were slightly reduced at levels equal to or greater than 500 ppm during the first generation only. Target organs were liver (adults) and kidney (adults and pups). The developmental and reproductive NOEL was 50 ppm, corresponding to a mean daily intake of 3.3 mg/kg clodinafop-propargyl.

Section 408 FFDCA 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. Base on the current toxicological data requirements, the database relative to pre- and post-natal effects for children is complete. Further, for clodinafop-propargyl, the NOEL of 0.32 mg/kg/day from the combined chronic/oncogenicity rat study, which was used to calculate the RfD, is already lower than the NOEL's of 40 and 25 mg/kg/day for the rat and rabbit developmental toxicity studies, respectively. Further, the developmental and reproductive NOEL of 3.3 mg/kg/day from the clodinafop-propargyl reproduction study is 10- times greater than the NOEL for the combined chronic/oncogenicity rat study. These data would indicate there is no additional sensitivity of infants and children to clodinafop-propargyl. Therefore, it is concluded that an additional uncertainty factor is not warranted to protect the health of infants and children from the use of clodinafop-propargyl.

Using the conservative exposure assumptions described above, it is concluded that the percentage of the RfD that will be utilized by aggregate exposure to residues of clodinafop-propargyl for the proposed use on wheat is 0.14% for nursing infants less than 1-year old, 0.34% for non-nursing infants, 1.05% for children 1-6 years old and 0.77% for children 7-12 years old. Therefore, based on the completeness and reliability of the toxicity data and the conservative nature of the exposure assessment, it is concluded that there is a reasonable certainty that no harm will result to infants and children from exposure to residues of clodinafop-propargyl.

F. International Tolerances

There are no Codex Alimentarius Commission (CODEX) maximum residue levels (MRLs) established for residues of clodinafop-propargyl in or on raw agricultural commodities. (Joanne Miller)

2. Office of IR-4

PP 8G4964

EPA has received a pesticide petition (PP 8G4964) from Office of IR-4, P.O. Box 231, New Brunswick, N.J. 08903-0321 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 temporary tolerance exemption based on no detectable residues in potatoes in 14 field trials and the limited nature of the EUP program or a temporary tolerance for residues of the insecticide spinosad in or on the raw agricultural commodity potatoes at 0.032 ppm which is 2x the limit of quantitation of the analytical method. 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.

A. Residue Chemistry

1. *Plant metabolism*. The metabolism of spinosad in plants (apples, cabbage, cotton, tomato, and turnip) and animals (goats and poultry) is adequately understood for the purposes of these tolerances. A rotational crop study showed no carryover of measurable spinosad related residues in representative test crops.

2. *Analytical method*. There is a practical method (HPLC with UV detection) for detecting (0.004 ppm) and measuring (0.01 ppm) levels of spinosad in or on food with a limit of detection that allows monitoring of food with residues at or above the levels set for these tolerances. The method has had a successful method tryout in the EPA's laboratories. Additionally, an Immunoassay has been developed.

3. *Magnitude of residues*. Magnitude of residue studies were conducted for potatoes at 14 sites in 7 States. No residues in potatoes were found in these studies with the lower limit of detection of 0.005 ppm.

B. Toxicological Profile

1. *Acute toxicity*. Spinosad has low acute toxicity. The rat oral LD_{50} is 3,738 mg/kg for males and >5,000 mg/kg for females, whereas the mouse oral LD_{50} is >5,000 mg/kg. The rabbit dermal LD_{50} is >2,000 mg/kg and the rat inhalation LC_{50} is >5.18 mg/l air. In addition, spinosad is not a skin sensitizer in guinea pigs and does not produce significant dermal or ocular irritation in rabbits. End use formulations of spinosad that are water based suspension concentrates have similar low acute toxicity profiles.

2. *Genotoxicty*. Short term assays for genotoxicity consisting of a bacterial reverse mutation assay (Ames test), an *in vitro* assay for cytogenetic damage using the Chinese hamster ovary cells, an *in vitro* mammalian gene mutation assay using mouse lymphoma cells, an *in vitro* assay for DNA damage and repair in rat hepatocytes, and an *in vivo* cytogenetic assay in the mouse bone marrow (micronucleus test) have been conducted with spinosad. These studies show a lack of genotoxicity.

3. *Reproductive and developmental toxicity*. Spinosad caused decreased body weights in maternal rats given 200 mg/kg/day by gavage highest dose tested (HDT). This was not accompanied by either embryo toxicity, fetal toxicity, or teratogenicity. The NOELs for maternal and fetal effects in rats were 50 and 200 mg/kg/day, respectively. A teratology study in rabbits showed that spinosad caused decreased body weight gain and a few abortions in maternal rabbits given 50 mg/kg/day HDT. Maternal toxicity was not accompanied by either embryo toxicity, fetal toxicity, or teratogenicity. The NOELs for maternal and fetal effects in rabbits were 10 and 50 mg/kg/day, respectively. The NOEL found for maternal and pup effects in a rat reproduction study was 10 mg/kg/day. Neonatal effects at 100 mg/kg/day HDT in the rat reproduction study were attributed to maternal toxicity.

4. *Subchronic toxicity*. Spinosad was evaluated in 13-week dietary studies and showed NOELs of 4.9 mg/kg/day in dogs, 6 mg/kg/day in mice, and 8.6 mg/kg/day in rats. No dermal irritation or systemic toxicity occurred in a 21-day repeated dose dermal toxicity study in rabbits given 1,000 mg/kg/day.

5. *Chronic toxicity*. Based on chronic testing with spinosad in the dog and the rat, the EPA has set a reference dose (RfD) of 0.0268 mg/kg/day for spinosad. The RfD has incorporated a 100-fold safety factor to the NOELs found in the chronic dog study. The NOELs shown in the dog chronic study were 2.68 and 2.72 mg/kg/day, respectively for male and female dogs. The NOELs shown in the rat chronic study were 2.4 and 3.0 mg/kg/day, respectively for male and female rats. Using the Guidelines for Carcinogen Risk Assessment published September 24, 1986 (51 FR 33992) (FRL–2984–1), it is proposed that spinosad be classified as Group E for

carcinogenicity (no evidence of carcinogenicity) based on the results of carcinogenicity studies in two species. There was no evidence of carcinogenicity in an 18-month mouse feeding study and a 24-month rat feeding study at all dosages tested. The NOELs shown in the mouse oncogenicity study were 11.4 and 13.8 mg/kg/day, respectively for male and female mice. The NOELs shown in the rat chronic/oncogenicity study were 2.4 and 3.0 mg/kg/day, respectively for male and female rats. A maximum tolerated dose was achieved at the top dosage level tested in both of these studies based on excessive mortality. Thus, the doses tested are adequate for identifying a cancer risk. Accordingly, a cancer risk assessment is not needed.

6. *Animal metabolism*. There were no major differences in the bioavailability, routes or rates of excretion, or metabolism of spinosyn A and spinosyn D following oral administration in rats. Urine and fecal excretions were almost completed in 48-hours post-dosing. In addition, the routes and rates of excretion were not affected by repeated administration.

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

8. *Neurotoxicity*. Spinosad did not cause neurotoxicity in rats in acute, subchronic, or chronic toxicity studies.

9. *Endocrine effects*. There is no evidence to suggest that spinosad has an effect on any endocrine system.

C. Aggregate Exposure

1. *Dietary exposure*. For purposes of assessing the potential dietary exposure from use of spinosad on cotton gin byproducts as well as from other existing or pending uses, a conservative estimate of aggregate exposure is determined by basing the TMRC on the proposed tolerance levels for spinosad and assuming that 100% of the cotton gin byproducts and other existing and pending crop uses grown in the U.S. were treated with spinosad. The TMRC is obtained by multiplying the tolerance residue levels by the consumption data which estimates the amount of crops and related foodstuffs consumed by various population subgroups. The use of a tolerance level and 100% of crop treated clearly results in an overestimate of human exposure and a safety determination for the use of spinosad on crops cited in this summary that is based on a conservative exposure assessment.

2. *Drinking water*. Another potential source of dietary exposure are residues in drinking water. Based on the available environmental studies conducted with spinosad wherein it's properties show little or no mobility in soil, there is no anticipated exposure to residues of spinosad in drinking water. In addition, there is no established Maximum Concentration Level (MCL) for residues of spinosad in drinking water.

3. *Non-dietary exposure*. Spinosad is currently registered for use on cotton with several crop registrations pending all of which involve applications of spinosad in the agriculture environment. Spinosad is also currently registered for use on turf and ornamentals at low rates of application (0.04 to 0.54 lb a.i. per acre). Thus, the potential for non-dietary exposure to the general population is not expected to be significant.

D. Cumulative Effects

The potential for cumulative effects of spinosad and other substances that have a common mechanism of toxicity is also considered. In terms of insect control, spinosad causes excitation of the insect nervous system, leading to involuntary muscle contractions, prostration with tremors, and finally paralysis. These effects are consistent with the activation of nicotinic acetylcholine receptors by a mechanism that is clearly novel and unique among known insecticidal compounds. Spinosad also has effects on the GABA receptor function that may contribute further to its insecticidal activity. Based on results found in tests with various mammalian species, spinosad appears to have a mechanism of toxicity like that of many amphiphilic cationic compounds. There is no reliable information to indicate that toxic effects produced by spinosad would be cumulative with those of any other pesticide chemical. Thus it is appropriate to consider only the potential risks of spinosad in an aggregate exposure assessment.

E. Safety Determination

1. *U.S. population*. Using the conservative exposure assumptions and the proposed RfD described above, the aggregate exposure to spinosad use on potatoes (using 0.032 ppm residue level) and other existing or pending crop uses will utilize 20.8% of the RfD for the U.S. population. No contribution to animal feed from potato was utilized in this analysis due to the limited scope of the EUP. A more realistic estimate of dietary exposure and risk relative to a chronic toxicity endpoint is obtained if average (anticipated) residue values from field trials are used. Inserting the average residue values in place of tolerance residue levels produces a more realistic, but still conservative risk assessment. Based on average or anticipated residues in a dietary risk analysis, the use of spinosad on potatoes and other existing or pending crop uses will utilize 4.6% of the RfD for the U.S. population. EPA generally has no concern for exposures below 100% of the RfD because the RfD represents the level at or below which daily aggregate dietary exposure over a lifetime will not pose appreciable risks to human health. Thus, it is clear that there is reasonable certainty that no harm will result from aggregate exposure to spinosad residues on potatoes and other existing or pending crop uses.

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

Section 408 FFDCA 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 for spinosad relative to pre- and post-natal effects for children is complete. Further, for spinosad, the NOELs in the dog chronic feeding study which was used to calculate the RfD (0.0268 mg/kg/day) are already lower than the NOELs from the developmental studies in rats and rabbits by a factor of more than 10-fold.

Concerning the reproduction study in rats, the pup effects shown at the HDT were attributed to maternal toxicity. Therefore, it is concluded that an additional uncertainty factor is not needed and that the RfD at 0.0268 mg/kg/day is appropriate for assessing risk to infants and children.

Using the conservative exposure assumptions previously described (tolerance level residues), the % RfD utilized by the aggregate exposure to residues of spinosad on potatoes and other existing or pending crop uses is 38.4% for children 1 to 6-years old, the most sensitive population subgroup. If average or anticipated residues are used

in the dietary risk analysis, the use of spinosad on these crops will utilize 11.3% of the RfD for children 1 to 6-years old. Thus, 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 spinosad residues on cotton gin byproducts and other existing or pending crop uses.

F. International Tolerances

There are no Codex maximum residue levels established for residues of spinosad on cotton gin byproducts or any other food or feed crop. (Beth Edwards).

[FR Doc. 98–15014 Filed 6–4–98; 8:45 am]

BILLING CODE 6560–50–F

EXPORT-IMPORT BANK OF THE UNITED STATES

Agency Information Collection Activities: Submission for OMB Review: Comment Request

AGENCY: Export-Import Bank of the United States.

ACTION: In accordance with the Paperwork Reduction Act of 1995, the Export-Import Bank of the United States (Ex-Im Bank) has submitted to the Office of Management and Budget (OMB) a request to review and approve a revision of a currently approved collection described below. A request for public comment was published in 63 FR, No. 59, 13437, March 27, 1998. No comments have been received.

SUMMARY: The Export-Import Bank of the United States (Ex-Im Bank) is soliciting comments from members of the public concerning the proposed collection of information to: (1) Evaluate whether the proposed collection is necessary for the proper performance of the functions of the agency, including whether the information will have practical utility; (2) evaluate the accuracy of the agency's estimate of the burden of the proposed collection of information; (3) enhance the quality, utility, and clarity of the information to be collected; and (4) minimize the burden of collection of information for those who are to respond; including through the use of appropriate automated collection techniques or other forms of information technology, e.g., permitting electronic submission of responses.

DATES: Interested persons are invited to submit comments on or before July 6, 1998.

ADDRESSES: Comments and recommendations concerning the submission should be sent to the OMB Desk Officer for Ex-Im Bank at the Office of Management and Budget, Information and Regulatory Affairs New Executive Office Building, Washington, DC 20503, (202) 395–7340.

FOR FURTHER INFORMATION CONTACT: Copies of these submissions and any additional information may be obtained from Dan Garcia, Export-Import Bank of the United States, 811 Vermont Ave., NW., Washington, DC 20571, (202) 565–3335.

SUPPLEMENTARY INFORMATION:

Abstract: OMB 3048–0005: Two applications fall under this collection. EIB–95–9 is the Ex-Im Bank Letter of Interest Application Form and EIB–95–10 is the Ex-Im Bank Preliminary Commitment and Final Commitment Application Form. There are no changes to either EIB–95–9 or EIB–95–10 other than a three-year extension of the expiration date.

Burden Statement Summary

Type of request: Extension of expiration date.

OMB Number: 3048–0005.

Form Number: EIB–95–9 and EIB–95–10.

Title: EIB–96–9—Ex-Im Bank Letter of Interest Application Form and EIB–95–10—EX-Im Bank Preliminary Commitment and Final Commitment Application Form.

Frequency of Use: Submission of Applications.

Respondents: Any U.S. or foreign bank, other financial institution, other responsible party including the exporter or creditworthy borrowers in a country eligible for Ex-Im Bank assistance.

Estimated total number of annual responses: EIB–95–9: 900, EIB–95–10: 550.

Estimated total number of hours needed to fill out the form: EIB–95–9: 20 minutes, EIB–95–10: 1 hour.

Dated: June 3, 1998.

Dan Garcia,

Agency Clearance Officer.

[FR Doc. 98–15167 Filed 6–4–98; 8:45 am]

BILLING CODE 6690–01–M

FEDERAL COMMUNICATIONS COMMISSION

FCC To Hold Open Commission Meeting Tuesday, June 9, 1998

June 2, 1998.

The Federal Communications Commission will hold an Open Meeting on the subjects listed below on Tuesday, June 9, 1998, which is scheduled to commence at 3:00 p.m. in Room 856, at 1919 M Street, NW., Washington, D.C.

Item No.	Bureau	Subject
1	Common Carrier	Title: Federal-State Joint Board on Universal Service (CC Docket No. 96–45); and Access Charge Reform (CC Docket No. 96–262). Summary: The Commission will consider action concerning proposals to ensure the accuracy and completeness of billing disclosures made by telecommunications carriers.
2	Common Carrier	Title: Federal-State joint Board on Universal Service (CC Docket No. 96–45). Summary: The Commission will consider action concerning the collection levels for the schools and libraries and rural health care universal service support mechanisms for the third and fourth quarters of 1998.
3	Common Carrier	Title: Federal-State Joint Board on Universal Service (CC Docket No. 96–45); Access Charge Reform (CC Docket No. 96–262); and Petition of Southwestern Bell Telephone Company, Pacific Bell, and Nevada Bell for Waiver of Sections 61.44–45 of the Commission's Rules (CCB/CPD 98–19). Summary: The Commission will consider action concerning issues related to local exchange carrier recovery of universal service contribution obligations.

Additional information concerning this meeting may be obtained from Maureen Peratino or David Fiske, Office of Public Affairs, telephone number (202) 418–0500; TTY (202) 418–2555.

Copies of materials adopted at this meeting can be purchased from the FCC's duplicating contractor, International Transcription Services, Inc. (ITS, Inc.) at (202) 857–3800; fax (202) 857–3805 and 857–3184; or TTY